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OF

OPHTHALMOLOGY

EDITED IN ENGLISH AND GERMAN

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NOTES AND CORRECTIONS.

I. CORRECTION.—In the "progress of ophthalmology," Vol. viii, No. 1, the notice, number twenty, of Dr. B. Joy Jeffries' first pamphlet on color-blindness is misleading. The examinations referred to as "not very accurate" were not made by him, but by the Sanitary Commission at the end of the Slavery Rebellion. Dr. Jeffries reported having at that time tested 611 persons, and finding 30 color-blind. Since then, as is well known, he has reported in his work on color-blindness his tests of some twenty thousand.

II. NOTE TO ARTICLE ON MELANOTIC EPITHELIOMA UPON THE FRONT OF THE EYE, p. 145, etc. By HENRY D. NOYES, M.D. A case of Dr. Hirschberg's, entitled, "Melano carcinoma polyposum precorneale" (see *Virchow's Archiv.*, Bd. 51, p. 515, 1870) was omitted from the list of cases of which I made use. I have to thank Dr. Hirschberg for calling my attention to it, and regret that it was overlooked. The case is most completely described, and strongly resembles that which forms the principal topic of my article.

New York, Nov. 20, 1879.

I am furthermore indebted to Dr. H. Knapp for pointing out two extremely valuable and pertinent cases, published by him in *Graefe's Archiv für Ophthalmologie*, vol. xiv., part 1, p. 280–284, 1868. Both cases had an auspicious result after having been submitted to operation.

III. NOTE TO THE PAPER ON CALCAREOUS FILM OF THE CORNEA, p. 293, ETC. BY E. NETTLESHIP.—Dr. Sellerbeck has quite lately recorded an interesting series of ten cases of this peculiar corneal opacity. Two of the ten appear to have been primary; seven occurred in eyes blind or defective from other causes (glaucoma, injury, sympathetic iritis, leucoma), and one deviated from the typical form. The author does not agree with von Graefe that when the change occurs on an otherwise healthy eye, *i.e.*, is primary, it predisposes to glaucoma. The opacity he finds, in common with previous observers, to be due to the presence of calcareous and magnesian granules under the epithelium, but the cause of the change is unknown. The hourly use of a five per cent. solution of hydrochloric acid is favorably spoken of, and iridectomy is preferred to scraping off the opacity. (*Hirschberg's Central-blatt.*, 1879, p. 159, abstract from *Charité Annalen*, 1879, p. 408–488, with woodcuts.)

IV. ERRATA.—Page 251, line 18, from below read *evert* instead of invert. Page 258, line 7, from below, read *flabby* instead of shabby. In fig. 257 the drawing of the threads is inacurate. The figure is reproduced in correct execution at page 406.

PREFACE.

THE ARCHIVES OF OPHTHALMOLOGY are the continuation of the ophthalmological part, and the Archives of Otology the continuation of the otological part of the *Archives of Ophthalmology and Otology*. This division has been made, both for the convenience of many readers, who practice either ophthalmic or aural surgery exclusively, and for the sake of doing justice to the growing importance of the literature in both departments. The English and the German edition have hitherto contained the same original papers, either in the language of the authors or in faithful translations. This plan will be essentially continued; but if the contributions become too numerous, some papers will be abridged in the translation. The editors will take care that by this condensation as little as possible of the author's investigation and thought is lost. If a paper is not translated *in extenso*, it will be so stated, in order to admit of reference to the original, should controversies of priority occur.

The review of the current literature, hitherto contained in the English edition only and consisting of selections, will in future appear in both editions and constitute a systematic and complete, though concise report on the progress of Ophthalmology.

Profiting by their past experience and relying on a great number of prominent men as regular contributors, the editors feel assured of being able to furnish to their readers a *good journal*, presenting the actual state and the progress of ophthalmic science and art in all their branches. In their selections the editors will give preference to those original papers in which speculation and theory are subordinate to direct observation and fact. Their greatest gratification will be to hear their readers continue to say: "What we read in your journal, we can make use of."

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ARCHIVES OF OPHTHALMOLOGY.

ON THE DEVELOPMENT OF THE CORNEA AND ANTERIOR CHAMBER.

By DR. W. C. AYRES, of New Orleans.
(From the Path. Laboratory in Heidelberg.)

See Plates I. and II.

AMONG the writings of the earlier authors, we find but little on the development of the cornea and anterior chamber, in fact not until Remak,[1] Koelliker,[2] Babuchin,[3] Ritter[4] and Hensen.[5]

Later we have special works, by Schenk,[6] Kessler,[7] Manz,[8] J. Arnold,[9] W. Müller[10] and Lieberkühn.

Inasmuch as the recent labors of Manz, Lieburkühn, J. Arnold, W. Müller and Kessler contain all the material necessary for the discussion of the various ideas as to the development of the cornea, both as a whole and in its details, it will be sufficient to speak of the differences which exist between the two prevailing theories upon this point.

Most of the above authors agree that, at least in the mammalia, before the receding of the lens, there is always between the *primary ocular vesicle* and the ectoderma, a layer of tissue which contains *cells*, and which is continuous on both sides with the *mesoderma*.

They further agree that this tissue is pushed in before the lens as it sinks into the secondary ocular vesicle, and envelopes it in its whole circumference in a closed sac, and

[1] Remak. Untersuch. über die Entwickelung der Wirbelthiere. 1855.
[2] Koelliker. Entwickelungsgeschichte des Menschen und der höheren Thiere, 1861.
[3] Babuchin. Beiträge zur Entwick. der Retina; Würzburg, naturwiss. Zeitschr, in Bd. IV.–V. und Stricker's Histologie. Bd. II., 1872.

that the layer of tissue which lies between the ectoderma and lens, goes to form the connective tissue portions of the *cornea.*

Koelliker was the first to point out the existence of this layer of tissue between the primary ocular vesicle and ectoderma, and to show that it was pushed in by the *lens* as it receded from the surface, and Zernoff, Lieberkühn, Arnold, W. Müller, etc., confirmed his conclusion, and stated further that this tissue contains *cells* and *nuclei* at every stage of its development.

The difficulty of accepting these views as to the first stage of development of the cornea, lies in the fact, that, before and after the receding of the lens, there is always immediately under the *ectoderma* a layer of tissue, which is in continuation with the *mesoderma*, and which always *contains cells.*

Some authors, and especially Kessler, deny, on the other hand, the existence of this layer of tissue. According to them, the cornea is in the beginning a *perfectly structureless* secretion from the ectoderma, and later it derives its cells from the sides by a peculiar process, which he calls cell migration, and therefore the tunica propria is a product of the ectoderma, which derives its cells from without, and therefore they are not developed "*in loco.*"

Were this statement correct, we would have here to deal with a most peculiar mode of development. That the cells of a connective tissue membrane in an embryonic condition must migrate into it from without, and even into an inter-

[4] Ritter. Zur histologisch. Entwick. des Auges; Arch. f. Ophthal. Bd. X. 1864.

[5] Hensen. Ueber den Bau des Schnecken-Auges. Arch. f. mikroskop. Anatomie. Bd. II., 1866.

[6] Schenk. Zur Entwickelungsgeschichte des Auges; Wiener akademische Sitzungs-Berichte math. naturw. Cl. Abtl. 2, Bd. 55, 1867. u. Lehrbuch d. Vergleichenden Embryologie, Wien 1874.

[7] Kessler. Ueber d. Entwickelung des Auges. Dorpat Dissertat., 1871, und Entwickel. des Auges der Wirbelthiere, 1877.

[8] Manz. Entwickelungsgeschichte des menschlichen Auges. Graefe u. Sämisch. Handbuch d. gesammten Augenheilk., 1875.

[9] J. Arnold. Beiträge zur Entwickelungsgeschichte des Auges, 1874.

[10] W. Müller. Ueber d. Stammesentwickelung des Sehorgans, Beiträge zur Anatomie u. Physiologie als Festgabe fur Ludwig, 1875.

[11] Lieberkühn. Ueber das Auge des Wirbelthierembryo, 1872, und Sitzungberichte d. Marburg. Gesellschaft. fur Naturwissenschaften, 1877.

cellular substance which has been the product of epithelial elements, were indeed a process so opposed to all established laws of histology, that it was this particular point which led me to the study of the subject under consideration.

Before presenting the results of my own investigation, I would only remark that all of my specimens were derived from the embryo of the *ox*, and a complete series from 6 *mm.* long to as large as desired, were examined in the course of the investigations, and I have chosen this particular animal also on account of the great similarity in process of development between the eyes of its embryos and those of man.

It is of course understood that only *well* hardened specimens were employed, and I agree with Lieberkühn in seeing no advantage in using *osmic acid.* The hardening in Müller's fluid and absolute alcohol, when carefully done, leaves nothing to be desired. As coloring materials I used carmine, hœmatoxyline and picro-carmine. I must also highly recommend the process of first staining the well hardened specimen; then extracting the water by alcohol; then placing it in oil of cloves; then in melted cocoa butter, and cutting specimens after the setting of the butter by cooling. Detachments of the ectoderma and shifting of the parts are thus much more surely avoided.

In the presentation of the results of my own investigations, I will begin with embryos of from six to seven *mm.* (6–7 *mm.*) long. The primary ocular vesicle appeared in such specimens of different sizes, which condition, however, is to be explained, first by the fact that the embryos were not always of the very same age, and secondly by the height of the plane through which the vesicles were cut.

When the distance of the anterior border of the vesicle from the ectoderma was great, there was always found between these two quite a thick layer of tissue, which contained *many cells*, and here and there *blood-vessels.*

In those cases in which the two had approached very near to one another, so that only a small space was left, the tissue between was of course thinner, and the cells fewer in number, but in no case did the vesicle and the ectoderma

come so close together that it was impossible to detect the intervening tissue, and in no case did this intervening tissue appear free from cells.

If the staining were not successful, then the cells were sometime difficult to see, but in specimens colored with hæmatoxyline not only could the cells be distinguished but also the protoplasm and the nuclei.

The connection between the tissue layer and the mesoderma, and their mutual dependence, could always be demonstrated.

My work therefore agrees with Koelliker, Zernoff, Lieberkühn, Arnold, W. Müller, when they say that this tissue always exists, and that it *always contains cells*, whereas it is in direct opposition to Kessler, when he insists to the contrary. Kessler derives his idea, in all probability, from the fact that the nearer the vesicle approaches the ectoderma, the thinner becomes this membrane and the fewer cells it contains, and it is true that at a certain time they are so few, that they are hard to demonstrate even with high powers. For instance, in certain animals, as in the smaller mammalia, this zone may become so thin that it may easily escape observation, but in the embryo of the ox it always has a certain thickness. In the case of the chick, I have seen myself how uncertain the question is, but I believe that W. Müller has even been able to demonstrate its presence even there. Nevertheless, in my opinion, the eye of the calf embryo is the proper object for this study.

In a later stage of the development, or that at which the lens begins to recede (embryos 9–12 *mm.* long), this zone of tissue between the *ectoderma* and *primary ocular vesicle* is pushed in along with it, as it hollows out the *primary* to form the *secondary vesicle*, and at every stage of the receding of the lens, this zone is still to be seen as a semi-transparent tissue containing cells. After the lens becomes detached from the ectoderma, it closes in and appears between the lens and epithelium, and is continuous on both sides with the mesoderma, and in the interior with the tissue surrounding the lens. Its appearance is even different in animals of the same age, but more so if the sections are not in the same

plane. In those sections which are either above or below the middle plane of the eye, this zone, between the epithelium and lens, is quite thick, and is very rich in round cells and blood-vessels. The nearer the sections come to the axis of the eye the thinner of course becomes this zone of tissue, and the fewer cells we find. They are flatter and more spindle-shaped or stellate, and the vessels also are fewer in number. By a superficial examination this membrane appears sometimes to be homogeneous, but by a more careful study, and especially with well-stained preparations, the existence of cells can always be demonstrated. The distribution of these cells is not at all regular, generally they are fewer in number in the middle and more numerous towards the sides. They are also irregularly distributed in the other direction, that is anteriorly and posteriorly, so much so, that often they are more numerous in the middle, and sometimes more numerous just under the epithelium. A homogeneous layer or zone is at this time not to be found. (Compare Fig. 1).

In embryos from 15–17 *mms.* long, this tissue is thicker and much richer in cells. At this stage of development I have examined an entire series of preparations, stained both with carmine and with hæmatoxyline, and the cells can be very distinctly seen. These are round, sometimes spindle-shaped, sometimes even stellate in form, and have an exceedingly well-developed cell-body, with round nuclei. At this period commences a peculiar arrangement of the cells, so that immediately under the epithelium they become very few in number, or are entirely wanting, and here the tissue has a more homogeneous appearance (compare Fig. 2). In general, this clear tissue seems to be identical with that which lies behind the lens, and with the mesoderma, and they are all mutually dependent and directly continuous with each other. Their vascular arrangement is also not essentially different.

The principal result of the above investigations is to lay stress upon the fact that immediately after the complete recession of the lens there appears a tissue between it and the epithelium, which always contains cells, in varying number

and arrangement, and blood-vessels. As already stated, the authors on this subject disagree so far that some (as Zernoff, Lieberkühn, Arnold, W. Müller, and others) assume that the cornea first makes its appearance as a tissue, which has cells and nuclei, whereas others (as Kessler) describe it as being in the beginning perfectly homogeneous. First, I would remark, that if immediately after the receding of the lens, that tissue which we find between it and the epithelium is the corneal tissue, then, from my investigations, it is *certainly not structureless*, but contains, at all times, *cells* and *nuclei*, but that this tissue is in no way to be distinguished from that which we find behind the lens and on both sides of it. It is indeed easy to understand Kessler's idea, for even in the case of the calf embryo, sometimes the cells are extremely difficult to see, and they may have been much more so in the material which he seems principally to have used in his examination. I must also state that the homogeneous zone under the epithelium described by Kessler, has made its appearance much later than he states, according to my investigations. I cannot agree with him that the zone belongs to the ectoderma, for in every instance in which the epithelium was detached, this zone remained in connection with the cornea and not with the epithelium. Hence neither its structureless nature nor its more intimate connection with the ectoderma can be conceded. There is no mistaking its cells for those that belong to the *stilus* of the lens—they are of an entirely different form. Moreover, their relation to the intercellular substance, their presence in every stage of the development, and their distribution, are, in fact, wholly opposed to this view.

As to the question whether that tissue lying between the lens and the ectoderma is the commencement of the corneal development or not, it may be remarked, that later the cornea certainly appears in this spot, and is continually developed from it; but at this time there is no appearance of the peculiar arrangement and stratification which characterize the cornea proper. From the results to be described, it will be seen what changes here occur.

For instance, in the very next period (from 18–21 *mms.*, see Fig. 3) of the development, we find the distinguishing characteristic is the inauguration of a lamellar arrangement. While in embryos from 15 to 17 *mms.* only a slight homogeneous zone had made its appearance under the epithelium, in embryos of 18–21 *mms.* this zone is very plainly visible, and has reached a considerable thickness (Fig. 3).

Cells or nuclei have by this time disappeared. There has also been a change in the middle layers of the cornea. The cells are more numerous, closer together, and the intercellular substance between them is much less than before. The form of these cells is sometimes circular, sometimes fusiform, sometimes stellate. In the middle the cell-zone is always the thinnest, and growing broader towards the sides, presents a conical figure in section, whose base is at the side or margin of the cornea, and whose apex is in the centre.

This appearance is precisely the condition of affairs that Kessler describes as a zone of cells which have wandered from the mesoderma on the sides into a homogeneous intercellular substance, which has had its origin in the ectoderma. I have shown that I could not endorse Kessler in his statement that the intercellular substance comes from the ectoderma, because it always and at all times contains cells, and is at no time homogeneous, as Kessler describes; and further, because it stands in much closer relation to the *substantia propria cornea* than to the ectoderma; and, finally, because this zone appears much later, at a time when the development of the cornea has already begun, and INCREASES in thickness, instead of DECREASING, as it should if it belonged to the ectoderma, and the cells were wandering into it from the sides. Moreover, in this clear zone under the epithelium, nuclei and cells are demonstrable at a very early period; later they disappear, and thus the homogeneous character is produced.

The migration hypothesis of the author I must pass by, because I was not able to obtain any confirmation whatever as to its existence; but this conical figure described by him, in which the cells are arranged, admits, in my opinion, of a much simpler explanation, if we recall the well-known fact

that, after the receding of the lens, the first development of the cornea begins from the side, and that the further development of the cornea goes on in the same direction, that is, from the sides towards the middle. The whole process at that period has made upon me the impression of a continuous development of tissue, and I am convinced the substantia propria of the cornea is developed from that zone of tissue between the lens and ectoderma by an increase in the number of cells, and an exudation of intercellular substance "*in loco.*"

The later development and metamorphoses that the substantia propria experiences, only confirm my views. (21–24 *mm.* Compare Fig. 4.) The cornea has in this stage a certain appreciable thickness, and while in the beginning it is much thinner in the middle, later it reaches about the same thickness throughout. The homogeneous zone under the epithelium shows no great change. The corneal tissue itself, however, is markedly altered, and now contains numerous cells, both fusiform and stellate, with round and oval nuclei, embedded in a tolerably thick, finely-granular protoplasm. The intercellular substance shows a striated appearance, or even traces of lamellation.

The discussion has been heretofore as to the development of that homogeneous zone which appears immediately under the epithelium, and of the substantia propria, but some observations still remain to be made upon the origin of the membrane of Descemet, and what is inseperably connected with it, the anterior chamber.

In embryos from 22–24 *mms.* in length, the space between the substance of the cornea and the capsule of the lens is filled by a transparent vascular tissue, which contains round, spindle and star-shaped cells, embedded in a homogeneous, intercellular substance. It, in fact, is perfectly identical with that tissue which was there immediately after the receding of the lens into the secondary ocular vesicle. While, therefore, the middle layers are changed into the characteristic appearances of the substantia propria, the inner layer preserves its structure. Both portions of this tissue are still very closely connected, and gradually become completely

blended (Fig. 4). Later, however, as in embryos of 25–30 *mms.* in length, a change begins in the layer behind the substantia propria by the appearance of a narrow, bright stripe next the corneal substance, in which there are neither nuclei nor cells. In embryos from 22 *mms.* this stripe is present, is exceedingly fine, but becomes broader, and finally forms the membrane of Descemet. Whereas this membrane makes its appearance much later than the anterior homogeneous lamella (the membrane of Bowman), nevertheless they are developed in precisely the same manner.

Simultaneously with these processes, changes are occurring in the layer of tissue next the homogeneous membrane, the future membrane of Descemet.

The tissue becomes less dense, and in some places it is interrupted so that small holes can be seen, while the cells are arrranged upon the walls of these interspaces. The tissue in front of the lens, however, forms a membrane, the vessels of which are immediately continuous with those running laterally and behind the lens.

In embryos from 25–30 *mms.* long, we find between the cornea and the vascular membrane in front of the lens, a net work of tissue, whose meshes are lined with flat cells. The same arrangement is met with on the posterior surface of the membrane of Descemet, where these meshes are not inserted.

In the further development these become fewer, by the spaces becoming larger, and the cell-lining of the membrane of Descemet more complete. At certain periods, finally, we meet with only a few isolated meshes covered with cells. Towards the corneal margin the meshwork is finer, and covered with cells, the future "ligamentum pectinatum." The place at which the development of this network begins is sometimes in the middle, and sometimes on the sides.

The anterior chamber is therefore formed by a separation of the cornea from the tissue which lays immediately below it, and with which it before stood in perfect continuation.

The endothelium of Descemet, as well as the cellular covering on the meshes of the ligamentum pectinatum, also owe their existence to this peculiar process, and, according

to my view, do not migrate, as Kessler insists, but are formed "*in loco.*"

The above observations agree with those of Zernoff, Lieberkühn, Arnold and W. Müller.

Kessler assumes that the endothelium of Descemet is produced by a wandering in of cells from the sides along the posterior surface of the cornea, and are not produced "in loco." This view is contradicted by the above-described method of development of the anterior chamber. Besides, I have often found that the endothelium appears earlier in the middle than at the sides, and progressed as the separation took place. This condition of meshes, lined with cells and stretched through the anterior chamber, and the development of the ligamentum pectinatum connected with it are unexplained by the views of Kessler, whereas by the method just described everything is remarkably clear.

Finally, mention must be made of the possible persistence of these fibres, similar to what we sometimes find in a membrana papillaris perseverans. It would also throw some light on congenital synechiæ, and declare them to be simply anomalies in the fœtal development, whereas at present they can only be accounted for by a supposition of an intra uterine inflammation.

Explanation of Plates I. and II.

The letters are the same on all figures, and

(*A*) Always represents the ectoderma or the epithelium of the cornea.

(*B*) The mesoderma or the substantia propria cornea.

(*C*) Vascular tissue between the cornea and the lens.

(*D*) The lens.

(*E*) The anterior end of the secondary ocular vesicle.

NOTES ON THE OPERATIVE TREATMENT OF DETACHMENT OF THE RETINA.

By J. HIRSCHBERG, of Berlin.

Translated by Isidor Furst, of New York.

DETACHMENT of the retina, unfortunately, is an affection as frequent as it is deplorable. In seven years I have observed it in 113 cases among 22,500 patients, *i. e.*, about 0.5 per cent.;* but I was not so fortunate as to see even one single instance of spontaneous and permanent *cure*, as some of my colleagues have done. Even cases of persisting *improvement* were extremely rare.

I have resorted to A. v. Graefe's *puncture of the retina* but sparingly, yet, although there was no loss of eyes nor grave reaction,† the results did not even come up to those modest expectations which an operative treatment of an affection of that nature would seem to demand. A moderate improvement (fingers from 6 to 8 feet after S—$\frac{1}{60}$), and once even a flattening of the elevations of the retina could be demonstrated after three years, but a perfect cure of the detachment could not be obtained, and speedy relapses, even while the patient was still in the hospital or soon thereafter, generally impaired or destroyed the original result.

The last case which I operated was the following:

A man, æt. 27, had lost the left eye long ago, the right 3½

* Professor H. Cohn, in Breslau, had, among 20,000 eye-patients in the years 1866 to 1876, 191 cases, or nearly one per cent., of detachment of the retina.

† I have punctured the elevations of the retina from the choroid, as I have seen A. v. Graefe do in 1866–1868, and not from the vitreous, as recommended by him in 1860 (*Arch. f. Ophth.*, Bd. IX), and as adopted in nearly all the text-books.

months ago, by retinal detachment in myopia. Right, fingers at 1′, prominent detachment of the lower half of the retina. Left, S.=$\frac{1}{\infty}$, detachment and degeneration of the retina. July 10th, 1875, puncture of the retina, right. A few days after reads small print. Two days later a relapse has destroyed the result. July 18th, second puncture of the retina, with immediate improvement. Two days later the result is again destroyed by relapse. Patient had to be discharged uncured.

This thoroughly unsatisfactory case caused me to abandon the operation. My experience was that of most of my colleagues. This is evidenced in the best manner by the compilation of Prof. H. Cohn (D. med. W., 1877, No. 32). Puncture of the retina, as recommended by A. v. Graefe, was performed in 1874 in 28 clinics, only 8 times among 20,000 operations, and in 1875 in 41 clinics, only 12 times among 16,300 operations.

Not wishing to adopt drainage—the execution and results of which I had occasion to observe in v. Wecker's clinic in 1876—on principle, owing to the foreign body remaining in the eye, I hailed A. Graefe's* new recommendation of the old scleral puncture as an advance in the treatment of so grave an affection.

Thus far I had occasion to perform scleral puncture for detachment of the retina in ten cases.

Case I.—Marie R.,† æt. 36, myopic from childhood, has impaired sight of the right eye for three months. Right, fingers

* Regarding the literature of scleral puncture in the treatment of detachment of the retina, I may be very brief. J. Sichel, in 1859, has proved the curability of detachment of the retina by scleral puncture in Clinique Européenne No. 29. He performed the operation on a myopic man, aged 40, with a lance-shaped knife downward, and raised the sight, which was very weak, to $\frac{1}{100}$. According to this, we have to correct the quotation of A. v. Graefe (Arch. f. Ophth., Bd. IX, 2, p. 35), which has been reprinted by several authors. Kittel (Wiener allgem. med. Zeitschr., No. 23), in 1860, has operated on one case; v. Arlt, at the same time, on several (Operations-lehre, p. 371); Wecker, in 1869 (Jäger-Wecker, Traité), likewise recommended the operation. I witnessed one operation performed with the lance-shaped knife by Coccius, who informed me that he had proceeded in this way for years. Kries (Arch. f. Ophth., Bd. XXIII, 1, p. 239) reported in 1877 A. Graefe's cases and recommendations; Pufahl described two cases operated by me (Centralbl. f. A., December, 1877); Wolfe (The Lancet, 1878, No. 15) has described scleral puncture as a new operation.

† Concerning cases I and II, comp. Centralblatt für Augenheilkunde, December, 1877.

eccentrically at 1′ outward, outward-downward, and downward. Upper half of field of vision completely absent, including the point of fixation. Left, Sn. 2 at 6″, field of vision normal (inward, 55°; outward, 85°, etc.), fundus normal. Puncture of right sclera on September 19th, 1877. October 26th, 1877, Sn. 3½ : 5″; field of vision normal; retina adherent throughout; isolated small gray spots in the fundus. November 28th, 1877, no trace of retinal detachment demonstrable; the ophthalmoscope shows M. 7 D. The limits of the field of vision normal in all directions (48° above, etc.), even with the small lamp; limits of color perception normal; tension equals that of the healthy eye.

August 6th, 1878, ten months and a half after the operation, *perfect cure* persisting. A most careful examination fails to show a trace of detachment of the retina.

Here and there in the fundus, a small lighter or darker spot (alteration of pigment) may be seen, and inward-upward toward the equator a somewhat more extended speckled patch (so-called choroiditis or chorio-retinitis). The eye reads Sn. 2 at 5″ and has an entirely normal field of vision (corresponding to, upward, 50°; downward, 60°; inward, 55°; outward, 80°, of the diagonal meridians).

RESULT.—*A high degree of detachment of the lower half of the retina for about three months, fingers at one foot eccentrically. Scleral puncture. Perfect cure, persisting after the lapse of nearly a year.**

CASE II.—Mrs. D., æt. 59, has seen badly with the right eye since the winter of 1876–77. Incipient cataract on both sides; left with —1.5 D. Sn. 40 at 20′; with +2 D, Sn. 1½ at 10″. Field of vision complete; On. Right, fingers at 6″, +6 D. Sn. 5 at 6″ hesitatingly. Nearly the entire upper half of the field of vision was absent, almost up to the point of fixation; besides, the upper half of the lower inner quadrant is lacking. (Field of vision: upward to 3°; inward, 8°; outward, 85°; downward, 60°, etc.) T—½. Catar. cort. post., still inconsiderable; detachment of retina downward, plainly perceptible through the dilated pupil. The case was kept under observation for three months, and found unaltered in every respect. October 10th, 1877, scleral puncture below-outward, beyond the equator. October 15th, Sn. CC. : 12′; +6 D. Sn. 3½ at 5″. Field of vision normal (upward, 40°; before, 3°, etc.). T. n. Detachment of the retina *not* demonstrable after

* Strange to say, *the first case was by far the best*, a fact which furnishes a strong argument against hasty deductions from a limited series of observations.

dilating the pupil. Condition remained the same several months. Then a limitation of the field of vision again gradually set in from within upward, central S. remaining unchanged (+6 D. : Sn. 2½ to 3 at 5″).

March 29th, 1878, limit of field of vision upward to 20°, perception of colors nearly normal. May 4th, 1878, limit of field of vision upward to 15°; June 13th, June 20th, July 8th, to 12°.

The field of vision, then, is better than before the operation, and finally almost stationary. The hemispherical detachment of the retina has not formed again. However, about 8 to 10 *mms.* from the papilla downward, a very narrow, whitish and barely prominent retinal fold may be seen, over which the vessels are passing. Beyond the fold the retina is firmly adherent, even to the extreme periphery. This fold may be explained on mechanical principles. The retina, after remaining abnormally distended for nine months by the detachment, had become too large for the fundus, and in reattaching folded. The limitation of the visual field *cannot* be fully explained by the formation of folds. If we assume that the fold interrupted conduction of the optic-nerve fibres, the defect of the visual field could begin no higher than 25° or 30° above the point of fixation. Certain cicatricial changes in the retina must have extended from the fold toward the papilla.

RESULT.—*An old case, unfavorable per se, existing for nine and stationary for three months, with defect of the visual field extending from above close to the point of fixation, relatively cured by puncture. The retina re-adheres, with the exception of a small fold. The field of vision at first becomes normal, later again limited from above, but almost stationary, and better than before the operation. S. remains permanently improved.*

CASE III.—Mr. S., æt. 41, always myopic, observed a veil in front of the right eye on April 11th, 1878; eleven days later, a high degree of visual disturbance.

April 25th, fingers barely at 1′; defect of field of vision downward, downward-outward, and downward-inward, extending above the point of fixation. Minute threads in vitreous. A large hemispherical retinal detachment bends from above over papilla and macula lutea. Left, Sn., On, My. of 10 D. Heurteloup, sudorifics, pressure bandage. The latter could not be borne on account of an old palpebral conjunctivitis, and had to be removed after one week. May 19th, 1878, right, fingers at 10′, +6 D. Sn. 3 at 4″. The print appears wavy. The field of vision extends up-

ward to 15°, thence to 40° there is indistinct perception of light. The detachment has settled downward, and now only persists in the lower half of the retina. The vision of the right eye slowly decreased, although the patient, being extremely careful, kept very quiet.

May 21st, 1878.—With +6 D. Sn. 8 at 6″. The upper half of the visual field almost completely absent, excepting only a central and a lateral triangular spot. June 1st, 1878, there remains in the upper half of the visual field only a narrow sector rising from the point of fixation. Correspondingly, in the inverted image, the upper half of the retina appears detached in two large elevations, between which a narrow depression remains, containing the optic nerve and macula. +6 D. Sn. 5 at 5″.

June 1st, July 13th and *14th.*—The defect of the visual fluid extends towards the lower half, increasing centrally and laterally from the point of fixation. The remainder of the visual field resembles a triangle with rounded corners, the base of which is downward, the apex above, including point of fixation and blind spot. The visual axis of patient being directed straight forward, the larger half of the artificially dilated pupil is occupied by the retina, which bulges especially toward the median line.

July 20th.—The narrow knife is inserted below inward, beyond the equator, is turned 45°, and left in this position for some seconds. Some drops escape; the scleral conjunctiva then fills like a vesicle; when the distention of the conjunctiva laterally has reached the horizontal meridian, the bandage was applied, although the retinal detachment had not disappeared, as a careful immediate examination showed. In the evening, the conjunctiva appeared less infiltrated; the visual field, tested by counting fingers, had extended upward; the retinal detachment had disappeared, two small peripheral wrinkles excepted.

July 21st, A. M.—Hardly any infiltration in the conjunctiva remaining. Downward, too, only a red reflex is visible with the ophthalmoscope.

July 27th.—Pupil moderately dilated by atropine. No trace of retinal detachment visible; but downward, in and in front of the retina, two or three rather copious hemorrhages. Bandage and dorsal decubitus continued.

July 30th.—With +6 D. Sn. 2. Field of vision above 56°, below 65°, outward 85°, inward 55°.

August 2d.—Limit for blue (square of 20 *mm.*) near 50°, red

50°. Green appears white, as also in the other eye. Patient states that his sense of color had always been imperfect.

August 4th.—With —4 D. Sn. LXX. at 12′. Hence My. less than left, and also less than before the retinal detachment. Patient was not discharged until three weeks after the operation.

Patient, a true type of a Government official, was a man of scrupulous obedience and perseverance. At home he remained quiet for weeks in a darkened room. A gastric nervous fever set in. *In the beginning of November a limitation of the visual field reappeared.*

September 2d, 1878.—The left eye recognizes Sn. CC. at 15′; with +6 D. Sn. 3 at 4″. The field of vision extends upward only to 15°, inward 12°, outward 30°, downward 60°, outward-downward to 80°. T. —½. The retina is detached from below, about up to the horizontal plane.

RESULT.—*Scleral puncture effects complete cure. This lasts one month, then a relapse follows.*

CASE IV.—Mr. H., some forty years old, has been affected with a high degree of myopia since his college days; has always worked much, and particularly hard during the last two or three weeks, when he observed a disturbance of sight in the right eye.

March 3d, 1877.—Left, Sn., On., highest degree of myopia. Right, the same degree of refraction, smallest print at 3 to 4″. Indistinctness of visual field below-inward. Above-outward there is a slight circumscribed retinal detachment, the margin of which is 4 to 6 P. distant from the optic-nerve entrance. Countless white glistening dots are visible in the slight detachment. General condition, aside from a moderate obesity, thoroughly satisfactory. Urine normal, etc. Iodide of potassium, moderate catharsis, rest in a darkened room, bandage, Heurteloup, in short, all "rational" treatment had no effect; on the contrary, the detachment increased and commenced to descend. Patient then travelled to Thuringen and indulged in energetic pedestrian exercise. This "irrational" procedure effected an improvement. A voyage to Switzerland followed later. On his return, August 11th, 1877, patient reported himself *cured.* The eye reads small print and counts fingers eccentrically in all directions. The retina adheres to the fundus, with the exception of some narrow peripheral and barely prominent folds directed straight downward.

Nowhere in the retina are there any white spots visible, although formerly these were so extraordinarily numerous near the centre.

On September 8th, 1877, without assignable cause, a relapse occurred; the eye reads only medium-sized print, has a defect of the field of vision upward, and shows directly downward a retinal detachment which has approached the optic nerve up to 3 P. Iodide of potassium. October, 1877, condition unchanged. No improvement having ensued up to January, 1878, operation is decided upon. The field of vision from above is absent almost to the point of fixation (upward 7°, above-inward and above-outward 10° each, outward 20°, inward 35°, downward 60°, below-inward 55°, below-outward 85°). Sc. pp.=$\frac{1}{40}$. The visual axis of the patient being directed straight forward, the lower half of the artificially dilated pupil appears occupied by the retinal detachment.

January 26th, 1878.—Puncture of the sclera below outward, during narcosis (by special request of the patient). Barely a drop of fluid prolapses, but nearly the entire lower half of the scleral conjunctiva presents a vesicular infiltration.

January 27th.—The œdema of the conjunctiva has disappeared. Patient states S. to be good. Only a trace of retinal detachment can be demonstrated downward. In the evening slight hemorrhage into the conjunctiva bulbi.

January 28th.—The eye is free from irritation. The time is easily told by the watch. No detachment of the retina demonstrable, but a slight hemorrhage in the vitreous downward. January 29th.—Vitreous clear, retina perfectly normal. January 30th.—Field of vision nearly complete, but downward a shallow *peripheral retinal detachment* may be demonstrated. Nevertheless, the field of vision is excellent (upward 58°, above outward 58°, outward 70°, below outward 80°, downward 60°, inward 60°, above inward 60°), and the eye reads Sn. 3½ with +6 D. The detachment soon became as before, but Sc. *remained better than before the operation.*

RESULT.—*In spite of long continuance of the detachment, scleral puncture effects complete adhesion of the retina and perfect restoration of function, but still in the hospital a relapse occurs.*

CASE V.—Augusta K., æt. 46, single, has always been myopic. Three weeks ago, after stooping very low, she noticed a red disc in front of the right eye; the disc remained, increased in size, and finally assumed a greenish tint.

Status præsens, December 10th, 1877.—*Detachment of the lower half of the retina.* Two prominent swellings (tense whitish ves-

icles), seperated by a deep depression, rise from below to near (3–4 mm.) the optic-nerve entrance.

An atrophic crescent exists at the lateral margin of the optic nerve, as also a circumscribed light spot near the centre of the retina. T.—1. Finger at 6–8′; —8 D. Sn. CC. at 12′; +6 D. Sn. 3 at 3½″. (Pupil artificially dilated.) The field of vision is limited from above (upward 25°, above outward 28°, outward 70°, below outward 70°, downward 60°, below inward 50°, inward 50°). Left, a high degree of myopia is found both functionally and objectively: Sn. 1½ at 4″; —8 D. Sn. LXX. at 15′.

Although vision is still tolerably fair, confident of the good results of scleral puncture, the operation is undertaken on December 10th, 1877. Patient sits upright. The lids are kept apart by a speculum. The conjunctiva bulbi is seized with the forceps below-inward at the equator, the globe slightly rotated on the visual axis, and the narrow knife inserted near the forceps. Only one drop of fluid escapes; but after withdrawal of the knife the corresponding quadrant of the sclera fills with thin watery liquid. The pupil contracts. Careful ophthalmoscopic examination immediately following the removal of the speculum shows the two swellings still persisting, though no longer as tense vesicles, but as flat roundish elevations with a reddish reflex. Bandage, rest in bed. In the evening the cornea is slightly wrinkled, no trace of retinal detachment visible.

December 11th, A. M.—The wrinkling of the cornea has disappeared, retinal detachment not demonstrable. Corresponding to the former external limit of the larger medial swelling, a very slight parallactic displacement of the retinal vessels may be seen in the inverted image, about equalling a difference in the level of ⅛ to ¼ *mm.* No fluid, however, rests behind the retina, but a thin diffuse layer of blood of considerable extent. The anterior limit of this layer of blood, directed toward the papilla, is formed by a brown streak, barely one P. broad and several P. long. This brown streak may correspond to the place of puncture.

December 15th.—Right, Sn. 2½ at 4″. Field of vision normal. Inward 50°, above-inward 50°, upward 48°, above-outward 65°, (outward 70°, etc.)

December 16th.—No retinal detachment demonstrable. In the night from 16th to 17th, patient—who at home performed the duties of a child's nurse—rose imprudently to attend to a crying child occupying an adjoining room with its mother.

December 17th.—The ophthalmoscope shows a recurrence of retinal detachment.

December 18th.—Some words of Sn. V., visual field absent from above to the horizontal line of demarcation. A narrow strip of indistinct perception extends from the point of fixation downward. Hemorrhagic flakes in the vitreous, which shows diffuse opacity. Extensive detachment of the retina downward. Decubitus, bandage.

December 19th.—The vitreous appears clearer. December 20th, a spontaneous rupture of the retina is plainly visible on the larger swelling. Flakes of blood adhere to the detached retina and float in the vitreous. December 21st, the retina is again adherent, the place of rupture (below-inward, some P. from the papilla, radially directed, 3 P. long, 1 P. broad) still plainly visible. Close to the margins of the rupture, the retina is covered with bloody filaments and flakes. Decubitus and bandage continued. Christmas, a second relapse of retinal hemorrhage into the vitreous, with slight detachment. On the evening of the same day, condition again improved. December 27th, detachment not demonstrable. December 31st, no detachment, but large flakes in the vitreous.

January 4th. 1878.—Flakes smaller. No detachment. Sn. 3 at 4″. Visual field nearly normal (inward 48°, above-inward 50°, upward 42°, above-outward 55°, outward 70°, below-outward 70°, downward 60°, below-inward 45°).

Patient, at her urgent request, is dismissed.

January 27th, 1878, a third relapse, communicated by letter, has occurred.

RESULT.—*Scleral puncture effects cure of a recent retinal detachment. Consequent upon traumatic influence, a relapse with intraocular hemorrhage occurs while still in the clinic. Spontaneous restoration, new relapse, etc.*

CASE VI.—Mr. S., æt. 28, myopic from childhood, writes a great deal, constantly wears glasses $-\frac{1}{16}$.

March 26th, 1878.—Right, Sn. 1½ at 8″, field of vision free, O. n.

Left, Sn. V. at 6″ with difficulty, defect of visual field from above to within 12° from point of fixation. The upper limit of the visual field runs nearly horizontal. Retinal detachment downward. Patient is treated for two weeks by pressure bandage in horizontal posture without effect. For the present dismissed by request.

The intended scleral puncture is *purposely* postponed, Case V. being borne in mind.

May 18th, 1878.—Left, with +6 D. some letters of Sn. 12. Fingers at 6′. The field of vision approaches a triangular shape, base downward; the rounded apex of the triangle rises only 5° above the point of fixation. May 19th, *scleral puncture*, without speculum, below outward, patient sitting upright. Bandage, recumbent position. In the evening there is œdema of the conjunctiva and pronounced wrinkling of the cornea. The upright image shows still a considerable retinal fold, detached downward. On the morning of the following day, this fold is unaltered, the conjunctiva nearly even, the cornea slightly wrinkled. Evening (May 20th), cornea smooth, no trace of retinal detachment demonstrable; but there is an extensive parenchymatous retinal hemorrhage.

May 21st.—No trace of retinal detachment visible through dilated pupil. Fingers counted eccentrically in all directions without hesitation. This condition continued up to May 25th. On the 26th, a rounded retinal detachment may be seen above-outward in the dilated pupil, by means of the upright image.

May 27th.—The detachment has again completely disappeared. May 28th, fingers counted across the whole length of the room. Reads Sn. 4 at 5″ with +6 D. Field of vision normal (outward 80°, above-outward 50°, upward 50°, above-inward 60°, inward 55°, below-inward 50°, downward 70°, below-outward 80°); no trace of retinal detachment can be demonstrated. May 29th, patient states on his morning visit that he has just had the misfortune to run against a chair with the left side of his nose. Considerable retinal detachment outward. Bandage, recumbent position.

June 10th.—Fingers at 1½′; field of vision limited to the lower outer quadrant, and thence not reaching to the point of fixation. Enormous retinal detachment, better seen with focal illumination. Coagula of blood in front of the detachment. T. —2. For the present patient discharged.

RESULT.—*Scleral puncture effects cure of a retinal detachment of several months' standing. Consequent upon traumatic influence, a relapse and impairment of function occur.*

CASE VII.—A. M., æt. 19, came under treatment February 19th 1878. His right eye had been injured two years ago while blasting rocks; the left had received a powerful blow, six days ago, from a jealous opponent at a dance hall.

Right, fingers at 4 to 5′. Visual field complete. Punctate corneal cicatrices; colobomatous defect of iris below inward; white capsular cataract; Tn.

Left, Sn. 30 at 15′; Sn. 3 at 6″. Defect of visual field extending from above to within 20° from the point of fixation. Lids of the left eye suffused with blood, but the globe itself is externally unchanged. Behind extensive flakes of vitreous, which, by focal illumination, appear blood-red; the ophthalmoscope shows a considerable retinal detachment downward. The left-sided retinal detachment remains unchanged after dorsal decubitus in a darkened room. Heurteloup's leech, iodide of potassium, etc.

Right, a considerable improvement of vision is obtained by iridectomy above-outward, thereby exposing a transparent pa.t of the lens (+10 D., Sn. C. at 15′, +15 D., Sn. 3 at 5″). Remarkable in this connection was an after hemorrhage, without assignable cause, into the anterior and vitreous chambers, and its slow absorption: even after two months, a red flake of blood, the size of a pin's head, could be seen unchanged close to the cicatrix, *behind* the delicate lens-capsule. As a defect of the visual field in the left eye, despite the complete rest of the patient, slowly increased from above, up to within 10° from the horizontal plane, scleral puncture was undertaken on July 12th, 1878, after five months' waiting.

It did not remove the retinal detachment, but considerably improved the function. The field of vision upward had gained a breadth of about 25°, and the color-fields were nearly normal, barring a slight horizontal depression from above. (Limit for white, above, 35°; for blue, 25°; for red, 20°; for green, 18°, etc. Square of 10 *mm.* on dark ground.)

RESULT.—*Traumatic retinal detachment, with slowly increasing contraction of visual field, of six months' standing. Scleral puncture does not remove the detachment, but improves the field of vision.*

CASES VIII AND IX.—H. W., post official, æt. 26, first presented himself August 16th, 1877. Wears glasses, —½ (inch measure). Right, Sn. 1½ at 2″ (pp), fingers at 3′. With —½, Sn. CC. at 15′, and Sn. 1½ at 2″. Left, Sn. 5 at 2″, fingers at 1′; with —½, fingers at 5′. Field of vision normal on both sides. There is on both sides the highest degree of myopia, staphyloma posticum, discoloration of fundus, also in centre; turbidity of vitreous, especially left. No trace of retinal detachment demonstrable even through dilated pupil. The strong glasses were taken

away and —¼ substituted; ordered Heurteloup's leech, darkened room, rest in bed, and resolvents. On August 24th, patient returned, complaining that letters appeared distorted in the right eye. Right, Sn. 1½ painfully at 2″; left, Sn. 5 at 2″. Right, a large sector-like defect of visual field upward, the point of which reaches to the point of fixation; throughout the rest of the visual field above the horizontal line there is indistinctness. Close above the papilla, in the inverted image, the vessels commence to turn black, and to project slightly forward; a flat and delicate though extended detachment of the lower half of the retina is present in the right eye. Although patient remained quiet on his back in a darkened room, yet on August 8th, 1877, the detachment of the right eye remained as before, and had recently occurred in the left; close above the left papilla, irregular folds and marked elevations may be seen. The deterioration rapidly increased. On November 18th, there is on both sides a narrow remnant of the visual field from the point of fixation downward, in which, on the right, fingers are recognized at 3′, on the left at 2′. Downward, the retina is pouch-like on both sides, covering the papilla; upward, the retina is flatter.

November 20th, left scleral puncture, below-outward. The yellow subretinal fluid, as usual, infiltrates the conjunctiva. S. immediately improved. November 21st, the œdema has disappeared, the tension of the globe greatly diminished. On ophthalmoscopic examination, shallow vertical folds in the cornea may be recognized. The retina still shows extensive detachment downward. In January, 1878, the right eye distinguishes fingers at 1′; the left, at 6′ eccentrically downward. The left eye, which, before the operation was worse than the right, is now decidedly better.

On February 20th, the retinal detachment on both sides may be seen with the naked eye, more distinctly right. Scleral puncture on the right eye, the knife being very slowly withdrawn, until the lower half of the conjunctiva is infiltrated with the transudation. Through the dilated pupil, the unarmed eye is enabled to recognize distinctly the recession of the retina during the efflux. Soon the right eye again counts fingers at 3′, the left at 10′. This condition remains absolutely stationary for eight months. Patient is again enabled to visit the dispensary alone. In the upper half there is a very shallow, almost capillary, detachment of the delicate retina from the red fundus; downward, the retina is widely detached in whitish elevations studded with brightly glistening

patches, evidently a state of connective-tissue degeneration, and perforated in many places; the ciliary part of the retina, likewise detached, appears like crape.

RESULT.—*Relatively early puncture of the sclera is advantageous, also in the malignant form of myopic retinal detachment which quickly grows total.*

CASE X.—A peasant, æt. 32, always very myopic—without hereditary predisposition or particular straining of the eye—blind on the right eye for two years, nearly blind on the left eye for six weeks, shows amaurosis left; right, fingers at 2′. Retinal detachment downward.

Right, scleral puncture with copious evacuation of the subretinal fluid, slightly outward, more largely into the conjunctiva. In the evening, the chemosis has disappeared, tells time by the watch without hesitation, counts fingers concentrically in all directions. No retinal detachment can be demonstrated. Week later, *status idem*.

A *result* cannot be communicated as yet.

If we review the above observations without prejudice, we must be impressed with the fact that we may resort to scleral puncture in nearly every case of retinal detachment, as an extremely simple and harmless procedure. As a rule, a *preliminary cure** will be obtained, leaving aside those unfortunate cases of total detachment of the retina.†

Relapses, however, from the nature of the affection, will form the rule rather than the exception; but the puncture may be repeated. Great caution and prolonged stay (three to four weeks) at the hospital, after the operation, are advisable. But all this is self-evident.

Less simple, however, is the question, which is the most suitable time for the operation, to exclude relapses and secure the best visual results? Von Arlt reports that he (about 1860) obtained good results from scleral puncture in *recent* cases of retinal detachment. A. v. Graefe maintained that it was best to await the process of spontaneous descent of the subretinal fluid, which would require about six weeks. This seems to me to be particularly indicated. In the first place, with the descent a spontaneous improvement of func-

* In A. v. Graefe's operation this was rather the exception.

† For those cases of tumor of the chorid which first manifest themselves in retinal detachment, the scleral puncture has a diagnostic value.

tion may set in, and in any case it is advisable *not* to operate during the period of greater fluctuation of visual power. Secondly, the result seems to have been particularly unstable in the operation of a recent case, although the detachment had occurred in a downward direction (Case V). Finally, it corresponds with the hypothesis that may be formed of the mechanism of ordinary (myopic) retinal detachment, to defer the operation until at least eight to ten weeks after the onset of the affection.

If an opportunity offers to make an ophthalmoscopic examination some days or weeks before the occurrence of a detachment of the retina, we generally find changes, the inflammatory character of which would hardly be questioned; such as dense, cloudy turbidity of the vitreous in front of the retina (probably connected with the detachment of the vitreous from the retina); or else grayish-black speckled patches, generally looked upon as an indication of chorioretinitis, with appended flakes or membranes projecting considerably into the vitreous. If we assume that, at least in a number of cases, the detachment is produced by an *effusion* of fluid behind the retina, we have good cause for waiting until the tendency to further effusions has to some degree subsided; but also, if the contraction of a pseudomembrane in the vitreous draws the retina from the fundus, it would be advisable to allow the subacute stage to pass by. Experience has demonstrated that the retina does not lose its perceptive power two to three, and even six to nine months after the onset of the affection. Of course, we must not delay too long (Case II), because the detached retina loses its elasticity, and its cicatricial contraction again slowly viciates the results gained by the operation.

A more extended experience will have to decide all these questions, and designate the best period for the repetition of the operation.

GLAUCOMA APHORISMS.*

By LUDWIG MAUTHNER.

Translated by James A. Spalding, M. D., Portland, Me.

B. SYMPTOMS AND NATURE OF GLAUCOMA.

I. *Excavation of the Optic Nerve.*

THOSE were the golden days of ophthalmology, when, twenty years ago, we gained an insight into one of the darkest of all diseases, glaucoma, by discovering the cardinal symptoms of increased intraocular tension and excavation of the optic nerve, and bringing them into the relation of cause and effect. Mackenzie indeed had stated twenty-five years before that a glaucomatous eye was hard, and had declared it probable that the retina became paralyzed by the pressure of the increased vitreous. But Mackenzie's idea was forgotten, and it came at last to be believed that the excavation of the nerve was a proof of the connection between the cause and the effect. The interruption of conduction in the optic nerve, followed later by blindness, was regarded as due to the pressing backward of the lamina cribrosa, and to the pressure upon the fibres of the optic nerve at the edge of the excavation.

But in these days we do not regard the connection to be so simple, and it seems to me quite worth the while to take up anew the discussion of several questions concerning the excavation of the optic nerve. Is the excavation a necessary symptom of glaucoma, or may it be absent when glaucoma is present? Is the so-called pressure-excavation really produced by pressure? What are the signs of the glaucomatous excavation? How is the excavation developed? Is a

* Continued from Vol. VII, page 242.

pressure-excavation capable of explaining satisfactorily the functional disturbances noticed in glaucoma?

V. Graefe said in 1862*: "The question might be raised whether an increase of intraocular tension can paralyze the functions of the retina without causing the optic nerve to yield. For a certain length of time this is certainly the case. But ultimately the diagnosis of the disease seems to be greatly aided by the fact that *every* increase of tension, influencing the conductibility of the retina, leads also to a *characteristic* excavation of the optic nerve."

Let us look somewhat more closely at this statement, and at once ask: What do we know of the *absence* of the excavation in glaucoma?

It would be trivial to point out that there is no excavation present when acute glaucoma breaks out in a previously healthy eye, or when in a case of fulminating glaucoma the sight is lost irredeemably. It would seem incredible that an excavation could have developed itself in the space of a few hours, or even indeed in the fraction of an hour. When the operation of iridectomy fails in acute glaucoma, and the eye, instead of regaining vision, remains or grows blind, we have certainly not to do with an especial anomaly of excavation. Rydel's case† of operation on an eye that was almost blind from acute glaucoma of three weeks' standing, having only weak quantitative perception of light, and where pain was removed and normal tension restored after an iridectomy but where vision was not improved, although there was no excavation, is not, although so frequently cited for this purpose, an example of glaucoma without an excavation, but only one more example to prove that the operation even in acute glaucoma does not always restore vision. The case of Madam M., reported in my previous paper, teaches us much more in this respect, for the operation, performed only six days after the outbreak of common acute glaucoma, did not restore sight, although it removed all the symptoms of glaucoma. The excavation was absent, but it would have developed itself in all these

* V. Graefe's Archives, VIII, 2, 280.

† V. Graefe's Archives, XVIII, 1, 57, 1872.

cases, just as after a certain period it develops itself in those eyes not submitted to an operation, but which might have regained their vision had the operation been performed at the right time. If in *such* eyes finally there were no excavation, *that* would be an unusual circumstance; but before proceeding to speak on that point, we have to touch upon a subject of universal importance—the state of the papilla in glaucoma hæmorrhagicum.

In cases where the increase of tension reaches its extreme degree, we ought, *a priori*, to expect, with the greatest certainty, a pressure excavation. But just here, the inclination is to deny the presence of a pressure excavation, or to look upon it as very exceptional. Only a few authors are willing to say that they have ever seen an excavation in glaucoma hæmorrhagicum. It seems to me to be of importance to come to a decision upon this question of principles. The cause of uncertainty on this point is due to the frequent impossibility of making an ophthalmoscopic examination at the time of the glaucomatous attack, and to the fact that, in order to relieve the pain the eye has often to be enucleated at an early period. It is not strange that we do not often find an excavation in the enucleated eye, for even the ophthalmoscope does not discover any in the cases where the fundus oculi can be examined.

There was no time for the excavation to develop itself. It must therefore be of the greatest interest to examine the fundus of an eye with glaucoma hæmorrhagicum *absolutum*, *i. e.*, at a later period in the course of an unrelieved hæmorrhagic glaucoma. The nature of the disease and the usual treatment resorted to (enucleation) make such cases very rare, but I have met with one.

Mr. Joseph E., æt. 63, a surgeon, presented himself to me at Innsbruck on September 29, 1871, with the following history: In 1860 his right eye became suddenly blind without any premonitory symptoms, while he was reading with his face turned towards a window where the very brilliant sunlight entered after reflection from a snow-drift. The patient hereafter guarded his eyes, wore dark spectacles, and the left eye remained perfect. The right eye improved so much in the course of a year, that he could once more read print unhesitatingly when using convex glasses.

Eleven years later, September, 1871, the left eye was attacked, and became blind in precisely the same way. After reading very closely for several evenings in succession by poor candle-light, a dark cloud came suddenly over the left eye, and still hinders vision. Now and then, at twilight, the patient is able to distinguish the contours of objects through this thick cloud.

On September 29, Dr. Fizia, who was then my assistant, made an examination, which yielded the following results: *RE*, $S = \frac{2}{30}$ barely; *LE*, fingers at 4′; *F*, limited upward, retinitis hæmorrhagica.

By the beginning of October (a few days after the appearance of the cloudiness), symptoms of subacute glaucoma developed themselves in the left eye, and increased by exacerbations to a violent acute glaucoma; this is remarkable, because, while in the right eye the central extravasation in the retina had been cured, the vision restored, and no relapses noticed in ten years, the same process in the left eye was followed by glaucoma.

In November, *RE*, $S = \frac{20}{30}$ nearly; *F* perfect, nothing abnormal in fundus oculi; *LE*, all the signs of a violent, acute glaucoma with violent pain; quantitative perception of light only when using the strong-light ophthalmoscope; no perception by diffused light; globe very hard, fundus oculi not visible.

This was the first case of hæmorrhagic glaucoma that had come under my care. I could not make up my mind to follow v. Graefe's advice and enucleate at once. November 10, I made a broad iridectomy upward, and the anterior chamber filled with blood. The pain disappeared instantly, so that the patient slept well the first night after the operation. The wound closed in forty-eight hours; the anterior chamber was restored, but remained full of blood, which, however, gradually diminished, while the eye remained much injected. After removing the pressure bandage, atropia and local blood-letting were resorted to. The patient was discharged sixteen days after the operation, with the eye only slightly injected, a trifling amount of blood in the bottom of the anterior chamber, and the fundus oculi still invisible. *Fingers were counted at* 8″, the pain had ceased, but the globe was still hard.

On January 31st, 1872, I saw the patient again. In the interval of four months he had had three violent attacks of pain, and he thought that they had been caused by over-exertion with his useful eye; the pain spread out over the forehead and temples towards the vertex and occiput. At present there is no pain.

The globe was pale, but after my long examination it showed a

slight injection in the ciliary region; *T* much increased (40 Monnik.) The wound is perfectly closed, free from iris and pigment, coloboma very broad, pupil very wide (suspicion on my part that the patient had been using atropia continuously), cornea clear, aqueous clear and free from blood, lens transparent, vitreous has a few floating opacities, fundus oculi visible with a strong illumination, although somewhat veiled. The papilla is destitute of vessels, the retinal vessels extremely thin, and some of them just after entering the retina seem broken off; there are a few small extravasations in the papilla and retina: *no trace of any excavation of the papilla;* no perception of light.

Since this last examination I have heard from the patient that the pain has returned for a short time on several occasions, but that the intervals of freedom were so long, that a second operation was never called for.

This case teaches us that there is a very peculiar state of things in glaucoma hæmorrhagicum, and even in the excavation of the optic nerve in general. Here, the glaucoma lasted four months, and with it an excessive increase in tension which evidently increased from time to time during the epochs of inflammation and pain; the retina was destroyed by hemorrhages, and vision was lost, but there was no trace of an excavation, nor did any of the vessels at the edge of the papilla, or upon it, show any bending or curvature.

Now, as we see from this case that the most destructive form of glaucoma, which often begins with the most excessive tension, does not seem to lead to excavation, so there are on the other hand cases of ordinary primary glaucoma in which an excavation is never developed, or appears at so late a stage that its recognition (if we wait for its appearance in order to be sure of our diagnosis of glaucoma) may indeed help our diagnosis but not our therapeutics. I pointed out in 1867,* "that we see cases in which the most pronounced symptoms of glaucomatous inflammation appear, disappear and re-appear, in which there is a marked increase in intraocular tension, and yet in which the symptoms of glaucomatous excavation are

* Ophthalmoscopie, p. 281.

not only never visible, but ultimately a simple atrophy of the optic nerve develops itself." By this I mean that *acute* glaucoma may become absolute, without the appearance of any excavation of the optic nerve.

I have since made similar observations, even in *chronic inflammatory*, as well as in *simple* glaucoma. We find an example of *chronic* inflammatory glaucoma (but, after all, the boundaries between acute and chronic inflammatory glaucoma are rather vague) in the case of George Knahler in whom glaucoma was cured by sclerotomy. When the media cleared up, there was no trace of an excavation, although the disease must certainly have lasted almost a year, and central vision had diminished to one-half. Neither did any excavation appear at a later period.

Much greater stress is to be laid upon the absence of an excavation in cases where no inflammatory symptoms have been present, for there is then no reason for making a diagnosis of *inflammatory* glaucoma. But when we find no excavation during a *pause of the inflammation* and none in glaucoma *simplex*, it may happen, and this too frequently, that glaucoma is not recognized. I have seen such cases repeatedly, and I will here say a few words about some that are still fresh in my memory.

The first case was that of a man about sixty years old, whose vision in both eyes was reduced to mere quantitative perception of light. Both anterior chambers were shallow, pupils wide and sluggish, eyeballs very hard. The diagnosis of glaucoma simplex seemed correct, but the mirror showed no trace of an excavation in the rather pale optic nerve, but did show complete absence of pigment epithelium. It would be a mistake in diagnosis, and also in therapeutics (vision being still present), if, under such circumstances, we should abandon our diagnosis of glaucoma simplex. Iridectomy upward in both eyes of this patient diminished the tension, but, as was to be expected, did not improve vision.

In 1873, while in Vienna, I saw Mr. B., æt. 30, who came to me complaining of blindness in his left eye, and asked my opinion as to its probable cause. I found the anterior chamber somewhat shallow, pupil somewhat dilated, slight ciliary injection and a few ciliary veins around the cornea; hence a glaucomatous condition of the eye. On comparing the tension with that of the

other eye, there could be no doubt of the presence of glaucoma. My wonder was not slight, for here was an intelligent man of the better class, who certainly had not delayed to seek surgical aid, and yet had become blind from glaucoma in a city like Vienna, without having an iridectomy done. My wonder increased when, on making an ophthalmoscopic examination, I found a most perfect glaucomatous excavation.

The puzzle was solved when I saw the notes of the surgeon who had been consulted. These showed that, at the time when the eye was already blind there was no excavation, and that consequently glaucoma was not diagnosed. But when the excavation did at last show itself fully developed, all hope of recovery of sight from an operation was lost. But as I learned at a later day, iridectomy, and finally, enucleation had to be performed, on account of violent inflammation which broke out in the blind eye. If I am not mistaken, I was also consulted at that time by my colleague Hock, in a case in which glaucoma could not be diagnosed from the appearance of the papilla, but we advised an iridectomy on account of the importance of the other symptoms.

A school-teacher, æt. 68, seeks advice about his left eye. R. E., shows glaucoma absolutum with cataract; L. E., a high degree of hypermetropia (of long standing), very shallow anterior chamber, pupil moderately wide, decided defect in vision, (the "intelligent" patient will not submit to a closer examination on this point) and distinct hardness. I am awaiting with certainty an excavation of the optic nerve, but there are no traces of it at present.

Other writers have also mentioned the absence of an excavation in glaucoma. V. Graefe, after speaking as early as 1858* of the "shallow" excavation to which glaucoma leads in eyes with sclerectasia, went somewhat more generally into the subject in 1869,† when he said: "Amongst the cases of subacute glaucoma, with occasional appearance of diffuse cloudiness in the aqueous, in the intervals of which the eyeball continues too hard, there are a few in which the excavation remains always disproportionally small to the anomaly of vision." The same holds good in a few cases of secondary glaucoma, and, even of glaucoma simplex. Thus he says: "I have seen eyes where reclination had been done, and eyes with cornea globosa, grow blind

* V. Graefe's Archiv., IV, 2, 153. † V. Graefe's Archiv., XV, 3, 232.

by degrees, with symptoms of decided increase of tension, in which there was indeed a *certain pressure excavation*, but it was entirely disproportional to the state of the vision. I recall, moreover, a few very old people, the tension of whose eyes had increased in a most marked degree (decidedly glaucomatous), the depth of whose anterior chamber and movements of whose pupils had evidently decreased, and who had lost their sight by degrees, accompanied by sharply-defined defects in the field of vision, with nothing to show for it in the papilla but *shallow depression* of its surface and an enlargement of the retinal veins."

In all these cases the excavation was "disproportionally slight;" it was "shallow;" there was a "certain sort" of pressure excavation, but still there was a real "pressure" excavation. V. Graefe mentions a single case only where it was absent.* This was the case of a joiner, æt. 52, who, having lost his phthisical and painless left eye three years before, had since been suffering from a weakness of vision in the right eye, which, as he said, had begun very slowly, but had increased rapidly during the last few weeks. The central *S* is moderately, the peripheral *S* extremely defective; there is a tendency to pericorneal injection, the pupil is very sluggish and not fully dilatable by mydriatics; the anterior chamber is somewhat shallow, and the tension *noticeably and constantly increased.* From all this v. Graefe thought that he would certainly find an excavation by ophthalmoscopic examination. *But instead of that, the level of the papilla and the relations of the vessels were perfectly normal:* and in the rest of the fundus oculi there were no morbid symptoms.

Mooren has had rather ripe experience in the apparent lack of diagnostic value of the excavation. He writes in 1874†: "There are a series of disturbances of vision in which, in spite of an increase in tension we cannot detect *a single trace of excavation*, but at the most only hints at arterial pulsation in the retina. Together with this marked diminution of central vision, we can always demonstrate a concentric, and now and then, a lateral contraction of the

* l. c. page 136. † Ophthalmologische Mittheilungen, pag 53.

field of vision after a long duration of the disease." In those cases also, which we may with Mooren call "glaucomatous choroiditis," "there is often merely a trace of excavation in spite of great hardness of the eyeball," while a great decrease in the acuteness of vision and peripheral contraction of the field are present. One case of "choroiditis" ended in a most perfect ectatic form in spite of an iridectomy, but although the increase of tension was enormous, the excavation of the optic nerve could by no means be called marked.

Cases of glaucoma simplex without excavation are not unknown to Schweigger:* "There are cases with palpable hardness of the eye, and symptoms of atrophic degeneration of the optic nerve, but without excavation, in which progressive blindness ensues, and may be improved by iridectomy."

The fact that the excavation is absent, or is perhaps only very "shallow," in processes which cannot be anything else than glaucoma, is important from several points of view. First of all, when we take this into consideration, glaucoma will be recognized more frequently than happens at present; for usually, unless we believe that we are sure of our diagnosis of glaucoma, we pay no attention to those cases without an excavation where we had expected as usual to find it, as if we had caught ourselves falling into a very great error. One result of this will be, that we shall employ in a series of cases that formerly resisted treatment, the correct treatment, an operative procedure, of the value of which in the circumstances under discussion I however possess no experience of my own, but, from the accounts of others, it seems indeed to be at least as successful as in the typical glaucoma. Schweigger says briefly, that iridectomy may remove progressive blindness coming on under such circumstances. Mooren, who says that such patients are very thankful for the operation of iridectomy on their eyes, although their vision improves very gradually, cites specially the case of a man, æt. 56, who could spell only J. 20, and whose field was contracted to a diameter of 3″, but who had

* Volkmann's Vortraege, No. 124, page 1038, 1877.

a normal field, and could read J. 5 with fluency a year after the iridectomy and the internal exhibition of silver nitrate.

But the absence of excavation in glaucoma has quite another significance. It leads to the question of causality between increase of tension and excavation. Eleven years ago, I argued thus:* "As the excavation in glaucoma is a pressure excavation, then the remarkable increased tension will at one time force back a lamina cribrosa of normal resistance, at another time (when the resistance of the sieve-like plate is below normal) the normal tension, or a tension a trifle above normal in the fluids of the eye will suffice to bring this about; while, if in case of increase of tension, the excavation of the optic nerve does not ensue, the lamina cribrosa itself does not yield to this increased tension, since it has an abnormally great capacity for resisting." V. Graefe says something very similar:† "Just as excavation of the optic nerve often develops itself very easily, owing to individual causes, with proportionally slight increase of tension; inversely, the development of an excavation may be resisted by existing anatomical conditions, and then we find far different consequences in the interior of the eye.

But when we consider that excavation does not, as a rule, develop itself in a decided form of glaucoma, viz., glaucoma hæmorrhagicum, in which the increase of tension reaches its highest degree; when we reflect that the excavation does not appear in many other cases of glaucoma, although the supposed greater resistance of the lamina cribrosa could not anywhere be proved, and that if the pressure alone were capable of bringing about such weighty results, many symptoms of excavation ought indeed to show themselves at the place of entrance of the optic nerve, even if the lamina cribrosa does not yield; then we shall be led to examine whether the typical "pressure" excavation is really such, and whether the appearances of the excavation, as well as the functional disturbances in glaucoma, can be explained by pressure upon the optic disc and retina.

The origin of the typical excavation, *i. e.*, one extending

* Ophthalmologie, I, 280. † Graefe's Archiv., XV, 3, 248, 1869.

to the edge of the papilla, by pressure, becomes constantly more unintelligible to me. The cross section of the optic nerve contains a certain number of nerve fibres. If now all of these, together with their interstitial tissue, waste away, we can comprehend the ophthalmoscopic picture of the glaucomatous excavation on the ground of a yielding of the lamina cribrosa. But how is it when the excavation has just developed itself, when there can be no question of an atrophy of the optic nerve fibres, and when the functions are still almost intact? The whole number of the nerve fibres must still cross over the edge of the papilla into the retina. If there is no excavation present, the whole surface of the papilla exhibits the cross sections of these fibres; with a *sharp-edged* physiological excavation, the edge of the papilla still standing on a level with the retina is the expression for the number of the fibres passing into the retina. It has been very difficult for me to comprehend how it is possible for this edge, which is often very narrow, to contain the same number of nerve fibres as in the unexcavated papilla, and how the cross section of a narrow ring at the periphery of a circle can contain the same number of nerve fibres as the cross section of the whole circle. The question whether a *physiological* excavation can extend to the edge of the papilla must, *a priori*, be called decidedly absurd, for let the fibres rising from the optic nerve into the retina press never so closely together, let all the interstitial tissue be never so much diminished, the nerve fibres when crossing into the retina must show a cross section at some spot, and in all excavations with sharp edges this must necessarily lie in the plane of the retina or rise above it. The only way in which I can imagine to myself the origin of a "pressure excavation" is that when the lamina cribrosa begins to yield, the mass of nerve fibres yield only in shallow grooves, and only when a high degree of atrophy of the nerve fibres has been reached, does the picture of a total excavation present itself; for so long as the pressure has spared even some fibres, so long must they rise over the edge of the papilla and form a boundary ring around its edge in case the excavation is not grooved or tun-

nel-shaped, but with sharp edges; or the excavation, when it involves the edge, may not be total, in which case there must be some spot at which, as I will assume, the fibres dislocated by pressure, but still capable of conduction, pass out over the edge of the papilla.

It is worthy of note that the results hitherto derived from pathological anatomy are at the first glance a source of more embarrassment than help. It is quite natural that eyes which have been blind for long periods from glaucoma should, when examined anatomically, show deep excavations with total disappearance of the nerve fibres, and it is necessary that the number of these examinations should be increased. But what is the state of the optic nerve in those cases in which the ophthalmoscope shows a total excavation, but where the function of the eye has not decreased at all, or only slightly? What has become of the whole convolution of nerve fibres which we no longer see rising up over the edge of the papilla? Is there in these cases also a pressure excavation that reaches quite up to the edge?

I will cite the oldest and newest anatomical discoveries on this point. H. Müller* described all the signs of most advanced glaucomatous excavation in the one blind eye of a patient, while in the other, which seems to have had some "glimmer of light," the cavity was tunnel-shaped, and the posterior part of the lamina cribrosa hardly at all displaced. From this description the ophthalmoscope would have found in a blind glaucomatous eye only a physiological excavation, and not even sharp-edged.

The latest accounts of the state of the optic nerve in glaucoma are those by Braily† and Schnabel.‡

Braily examined fifty-three cases of glaucoma, amongst which, however, as he notices with regret, there was no case of fresh glaucoma simplex. And what did Braily find? "The first effect of pressure appears in a yielding of the centre of the lamina cribrosa." Not until later does "a central funnel-shaped, ill-defined, small depression" develop itself in the optic disc, which advances towards the lamina,

* Graefe's Arch., IV, 2, 28, 1858.
† Ophth. Hospital Reports, IX, 2. Dec., 1877, page 207.
‡ These Archives, Vol. 7, 1, 12.

and later extends laterally, until at last the scleral ring of the optic nerve lies almost bare. Excavation with an *overhanging* edge is on the whole only reached after great pressure, although *as regards the total destruction of the nerve fibres*, the effect of increased and diminished pressure is the same. Not a single case, where the excavation showed undermined edges, had lasted less than two years; and in several cases with a history of seven and more years' duration, and with $T+1$, on enucleation, the cavity *was neither deep nor laterally undermined*, so that when T is but moderately increased, its continuance for years does not necessarily produce a "flask-shaped cup;" there is merely a *wider cavity*, whose *centre* is usually the *deepest* part. Braily suspects, from the results of this anatomical examination, "that very many of the so-called atrophic excavations, such as the ophthalmoscope shows, belong to a protracted subacute (?!) glaucoma simplex, in which the tension never rose distinctly above the normal, or else did not at the time of examination."

The author adds a sketch (sagittal section through the optic nerve) which represents the beginning of a glaucoma excavation, under the form of a central physiological cavity, but with the lamina much pushed back, and a second, the well-known ampulla depression, with total atrophy of the nerve fibres.

Schnabel's thirteen cases include seven which belong to blind eyes with a high degree of atrophy of the nerve fibres. It is natural that an examination should reveal the typical excavation in these cases. Since we are here looking simply for the anatomical relations of the newly-developed glaucomatous cavity, we do not consider eye No. 1 without excavaation, eye No. 3 with swollen papilla, and eye No. 8 with purulent inflammation of the uveal tract. We have left, therefore, of 13 eyes, only 2, Nos. 2 and 6. Eye No. 2, which had sight up to the time of the patient's death, shows, as Schnabel says, "a partial physiological excavation, while the lamina cribrosa has its normal form and situation." This discovery teaches us nothing, for during life the typical total excavation was wholly wanting; the only thing that it could

teach us was that it had nothing whatever to do with glaucoma. Eye No. 6 *has no ophthalmoscopic* picture; this would have been the only thing of interest about the case; but as the nerve fibres were not yet atrophied, the *anatomical* examination revealed only a *funnel-shaped, partial* excavation, in spite of the yielding of the lamina cribrosa.

From all this we are to learn *that no eye has ever yet been examined anatomically in which both an undeniable typical glaucoma excavation had been previously demonstrated by the ophthalmoscope, and preservation of the function of the eye, even in a diminished degree, had been ascertained. All anatomical examinations of glaucomatous eyes in which the layer of nerve fibres has been preserved, never give, in spite of the yielding of the lamina cribrosa, any other picture than that of a partial, funnel-shaped excavation, a picture from which it is impossible to explain, according to our present views, the ophthalmoscopic appearance of a "pressure" excavation extending to the margin of the disc, while the former general considerations render the results gained by the microscope self-evident.*

It is impossible, therefore, for the apparent picture of a "pressure" excavation to be produced in the beginning by pressure, for, in order to produce such a picture by pressure, the collective bundle of nerve fibres, or the larger part of them, must, in point of fact, be cut off close around the scleral ring; and, moreover, none, or only a very few, of the nerve fibres can cross over the edge of the disc, an assumption which is simply monstrous, when we consider that up to this time the functions of the central and peripheral parts of the retina have been wholly or almost entirely preserved.

In addition to this, we find that in the beginning of an excavation the pressure is frequently not increased. I have recently examined the left eye of a patient, in which there is the beginning of a pressure excavation, of which there was not the slightest sign a year ago when I saw the patient for a paresis of one of the muscles. The functional disturbance is extraordinary, but shows itself in transitory obscurations; central S is less than in RE, which has $S=\frac{6}{6}$, while with LE, a few letters of 6 are not seen at 6 *mm.* dis-

tance. Without glasses, the patient, who is 45 years old, reads with *RE*, J 2, with *LE*, J 3; *F* undisturbed. The well-known appearances of the vessels are very marked at the upper lateral edge of the left papilla. *T* is precisely the same in both eyes, and falls even below the physiological maximum. Would such a pressure produce a picture at one part of the edge of the disc, as if all the fibres, together with the vessels, were there cut off sharply, as if the quadrant of the disc were then pushed back, and the scleral ring pushed inward a trifle over it?

The fact that we cannot demonstrate an increase of tension in many cases of glaucomatous excavation is admitted by several ophthalmologists, leaving out of consideration those who, following Donder's theory of glaucoma simplex, have expressed such an opinion directly. But this excavation is nevertheless assumed to be a pressure excavation, and the disease to be glaucoma, because it happens that we find in one eye simple glaucomatous excavation without perceptible increase of tension, but in the other eye glaucoma with the excavation, with all the characteristic symptoms and also a distinct increase in tension. This conclusion does no honor to science. When I find a deep glaucomatous excavation in one eye *without any trace* of increase in tension, and in another eye a similar excavation developed with *distinct increase in hardness of the globe*, there is but one logical conclusion to be drawn from these facts, viz., that the excavation cannot be produced by pressure. But, in point of fact, what conclusion have we drawn? We have concluded that, *because* in one eye the pressure is increased and excavation visible, in the other eye, the pressure of which is notoriously not increased, but which also has an excavation, the pressure must be increased. Such a conclusion may be set up as a *dogma*, but the time has come when we should not further "sacrifice our intelligence" in *believing* such teachings.

Before going further, I desire to show how to my mind the picture of a total excavation due to pressure, when the functions of the eye are still preserved, can only be an apparent one. Some morbid process, which has attacked the

intraocular end of the optic nerve (but, as we shall see, not this only, and probably, also, not primarily) causes a diminished resistance (softening) of the lamina cribrosa, so that it yields even to normal pressure in the eye, just as the elongation of the axis in myopia is caused by normal pressure upon a sclera which has but little power of resistance. But at the same time, there is going on in the optic nerve an alteration which has the greatest resemblance to that in the lamina cribrosa, and leads to a softening, to a giving way of the supporting connective tissue. In this way, on the one hand, the fibres of the optic nerve become extraordinarily transparent, and on the other hand the vessels sink backward into the transparent head of the optic nerve. Thus it seems as if the latter were pressed close to the scleral ring, while the masses of nerve fibres covering them (for they cannot have disappeared if this function is preserved) cannot be seen directly with the mirror. In this way we can explain why the anatomist does not demonstrate a total excavation which he cannot find in eyes that have not become blind from glaucoma, but either no excavation at all, or only a *partial* one, in the end of the optic nerve discolored from exposure to the action of hardening fluids. This explanation seems very forced and I would, indeed, cheerfully welcome any other, but desire to call attention to certain ophthalmoscopical appearances that speak in its favor. In the first place, when we make anatomical examinations of hardened glaucomatous eyes, it is not so rare to find in the perfectly excavated hollow whose nerve fibres have disappeared, a newly formed, more or less homogeneous or striped mass, not a trace of which was to be seen when we made our ophthalmoscopic examination. In the same way a picture of the end of the optic nerve that has reached its highest degree of transparency, has hitherto escaped us. But this is not a novel idea. As long ago as 1861 v. Jäger called attention to *apparent* physiological excavations, where it is supposed that there is a hollow reaching quite to the lamina cribrosa, or even beyond it, but which is really the result of a remarkable transparency of the nerve fibres. I desire here to note that even here, in so far as the optic disc is concerned, we may have to do not

so much with an increased transparency of the nerve fibres that are usually very diaphanous, as with an abnormal deficiency of supporting tissue.

If my view is correct, we shall comprehend another phenomenon which must be known by every ophthalmologist. We often see a picture of total excavation with all the characteristic appearances in the large vessels, while one or more smaller vessels cross over the edge of the papilla and run some distance towards its centre without showing a trace of bending or curvature, and a careful examination shows that the vessel on the papilla (before sinking into its substance) lies, in point of fact, at about the same level as when outside the disc. If the situation of the greater vessels shows the situation of the layer of nerve fibres over which they run in a normal state, and if the surface of the optic nerve is sunken deeply backward, the course of these single vessels at almost their original level is incomprehensible. Has this vessel, which is undoubtedly retinal, lifted itself away from the upper surface of the papilla into the vitreous, while the surface itself has sunk backwards? I could not explain the phenomenon in any other way. But it explains itself, if we abandon the idea of a sinking backward of the mass of nerve fibres, and take it for granted that the vessels, deprived of their supporting tissue, have fallen backwards as a result of intraocular pressure. In such a case it would not be strange if a vessel whose surrounding connective tissue had suffered but a trifle, should at first keep its original position, nor would it be strange, since the morbid process does not at once affect the whole surface of the nerve, that these appearances of the vessels should first be seen at one spot on the edge, and that restitution is still possible at the very beginning of the disease.

Perhaps it will be asked why the bending of the vessels always begins at the edge of the disc. In answer, I would say that this indeed is not always the case, although, to be sure, my view explains the sinking of the vessel at the edge. If at the *beginning* of a presumptive glaucoma excavation we do not see the bending of the vessels at the edge, we do

not then know whether we have to do with a physiological or a glaucomatous excavation, and we seldom succeed in following up the process (Klein). But by careful examination of cases of advanced glaucoma, I have received undeniable evidence that even there the excavation does not extend to the very edge, but leaves a peripheric part of the disc intact. I have but little doubt, from my own experience, that other observers may also be deceived, mistaking "the edge of the papilla" for a "glaucomatous ring." After finding in several cases, by exact examination in the erect image, that the apparently typical picture of a total excavation, surrounded by a narrow ring, was nevertheless deceptive, I became convinced that it would be impossible to be free from error if we should examine only in the usual inverted image. The case of Andrew Praxmaner offers a striking example. Here we have a blind eye, a glaucoma absolutum with intercurrent inflammatory symptoms, and yet the excavation does not affect the whole surface of the papilla.

As to the action of intraocular pressure in the causation of the excavation, I am convinced that total atrophy of the nerve fibres would also gradually be developed in case the space occupied by the vitreous were a vacuum. But the atrophy is certainly increased by the normal, and still more by the increased intraocular pressure. Still, at the same time, the mass of nerve fibres can only decrease from the centre towards the periphery, and a *true* excavation only be produced by advancing from the centre towards the periphery, until at last in *complete* atrophy the cross section of the excavation will be denoted by the position of the vessels. When Brailey finds that a decidedly high pressure is necessary for the formation of a *cup-shaped* excavation of the *lamina cribrosa*, since an excavation with overhanging edges is never formed by moderate pressure, then I have nothing to reply from my point of view.

Besides this, there are other points to be considered. There have been several descriptions, and even representations, of peculiar convolutions of fine vessels in the depths of the excavation, and which have been thought due to an

increase of intraocular pressure. But since the pressure bears most especially upon the *edge* of the excavation, we assume a turgescence of the blood in the *veins* at the *edge* of the papilla; therefore the veins in the floor of the excavation hold less blood, so that there can be no question of an engorgement by turgescence of the veins in the floor of the excavation. Or are they distended arterial branches? These convolutions have no resemblance with distended arteries, but look much more like the formation of new vessels seen in "engorged papilla." I do not hesitate to regard these very singular vessels as new formations, developed during a morbid process in the head of the optic nerve, independent of any increase of pressure.

And what is the origin of the newly-formed tissue that sometimes covers the floor of old excavations? If pressure and pressure alone is active, if atrophy and atrophy alone is produced by pressure, I do not understand how there can be any new formation of vessels in the floor of the excavation.

Finally, we have to consider the "glaucomatous ring"—that very peculiar yellowish ring surrounding the glaucomatous optic nerve. Schweigger discovered by anatomical examinations that this ring is due to atrophy of the choroidal tissue. I do not doubt but that this may be the ultimate result, just as in spots recognized as exudations in the choroid we find at last an atrophy of the choroidal tissue. But still I doubt exceedingly whether the primary affection is an atrophy of the choroid. We often find this ring without a trace of remains of choroid, and, on the other hand, without a trace of the whitish-blue tint of the sclerotic. Then we see pictures where a part of the ring still shows its *uniform yellow* tinge, while another part looks just like a conus, *i. e.*, it bears remains of choroidal vessels upon a whitish ground. Finally we see, in cases of old glaucoma, that the optic nerve is no longer surrounded by the glaucomatous ring, but *encircled* by a "conus." And still more remarkable, we often find that some of the large vessels entering upon the optic nerve are cut short, not at the edge of the optic nerve, not even at the inner edge of the ring, *but at its outer*

edge. I have proved this fact conclusively in a few cases. I saw it last in an eye whose optic nerve I had given to Dr. Purtscher at Innsbruck to draw for quite another purpose. Dr. Purtscher then pointed out to me that an upper venous trunk was cut off sharply at the external edge of the ring. This was really the case. Amid the confused mass of vessels covering the floor of the excavation one was plainly to be regarded as the continuation of the recognized venous trunk, and into it, just at the outer edge of the ring, there opened a still smaller venous branch, crossing *over* the ring.

Besides this we must not overlook the fact that the ring is sometimes seen developed before the excavation. The case of Kurzemann, teaches us a great deal on this point. Both of his eyes showed the same state of the refracting media, and the same acuteness of central vision. In the right eye there was a pronounced total excavation, with a broad glaucomatous ring, while in the left eye, which at that time showed a somewhat higher tension than the right, the yellow ring was very distinctly marked, but the excavation was barely hinted at. The probability here is that there is really an exudation around the optic nerve, which, as it covers a few retinal vessels that evidently lie at some depth, seems to press forward from the choroid into the retina to a quite decided depth, and at first to be the cause of the yellow ring, while in *later* stages atrophy of the tissue ensues after resorption of the exudation, and hence the sclera appears as if covered with more or less remains of the choroid.

Now that I have expressed my views and experiences about the meaning of the glaucoma excavation, I desire to cast a rapid retrospect at analogous views of other ophthalmologists. For this purpose I will refer to but three works that appeared in 1876. V. Jäger,* partly in defence of his previously expressed opinions, explains "the total and steep-edged excavation of the optic nerve in connection with the rest of its characteristic appearances (the glaucomatous disease of the optic nerve)" by the development of the glaucomatous process in the region of

* Ergebnisse der Untersuchung mit dem Augenspiegel, pag. 23.

the scleral vessels, while, if the glaucomatous process remains confined to the region of the choroidal vessels, the glaucomatous disease of the optic nerve is wanting. The significance of the increase of intraocular pressure is, that it influences the size of the excavation of the optic nerve in proportion to its intensity and duration.

Schnabel* is of the opinion that the total excavation with steep walls is indeed produced in a large number of cases by the increase of pressure alone, but that it originates now and then from the united action of other causes. He supports this view from his own experience, thus: that the glaucomatous ring is sometimes developed to a very considerable breadth when the excavation is still scarcely recognizable, and that in secondary glaucoma (when, in Schnabel's opinion, the excavation is always a pure pressure effect), the glaucomatous ring is very seldom developed, while in the common glaucoma simplex it is only exceptionally absent. Then again, a point in favor of the view of an independent disease, is the circumstance that the excavation, even with a previous physiological cupping, always is formed at the *edge*, and finally a singular fact, that a perfectly characteristic glaucomatous excavation became developed in a case of acute glaucoma two months after a perfectly successful iridectomy, although the sight had remained wholly unchanged, and the pressure had not increased.

Klein† published a similar case later, only increase of tension and other glaucomatous symptoms were present. Klein also has seen the ring *before* the excavation, and he adds another most wonderful fact, that in one case the ring *disappeared* after the iridectomy. He has also followed up the development of glaucomatous excavations, and comes to the conclusion that it frequently proceeds in this way; that the most varying portions of the papilla, at the edge as well as in the centre, may become cupped one after another to the amount of a half of the disc or a quadrant, or even smaller sections. This state of things, as well as the circumstance that in the meanwhile the intraocular

* These Archives, V., 369. † V. Graefe's Archiv., XXII., 4, 157.

pressure is but rarely increased, while on the other hand, with evident increase of pressure, the excavation does not necessarily follow, gives rise to the opinion, when taken in connection with the formation of the glaucomatous ring on a sclero-choroiditic basis, that the "pressure" excavation cannot originate without a glaucomatous affection of the optic nerve.

While therefore, Schnabel is only a conditional supporter of v. Jäger as regards the glaucomatous affection of the optic nerve, Klein supports him unconditionally. As for myself, I stand wholly with v. Jäger and Klein, in so far as I cannot regard the total excavation as a pressure excavation, and for this reason among others, that, so long as the function is preserved, I do not, on the whole, regard it as a total excavation.

II.—The disturbance of function.

I believe that the chief opposition to the view that the glaucomatous excavation is a pressure excavation, is to be deduced from observations of the functional disturbances in glaucoma, the accurate consideration of which, however, does not permit us to coincide with v. Jäger's view, that in the affection which leads to glaucomatous excavation we have to do with an inflammatory process, which is seated exclusively within the circle of the scleral vessels.

V. Graefe,* as is well known, was a strong opponent of the significance of pressure in the production of functional disturbances, by direct action upon the nerve substance. In acute primary glaucoma, where the excavation does not yet play any *rôle*, he considers it an inadmissible view, that "the increase of pressure obstructs the nervous conduction as such." Here we have to do rather with an ischæmic paralysis of the retina, and probably too, with a simultaneous disturbance of nutrition in the retina,—"fragility, (softening?) of the retinal tissue,"—all of which is naturally caused by pressure. As regards the significance of the excavation of the optic nerve, v. Graefe is far from† opposing in all points the theory that the interruption of the conductibility

* V. Graefe's Archiv, XV, 3, 112.

† l. c. page 232.

of the fibres at the edge of the excavation, is the sole cause of the functual disturbance, a theory that really has become the basis of the later teachings in glaucoma, but he thinks that in certain cases a modification of it must be adopted. Then he begins to speak of glaucoma without, or with a disproportionally shallow excavation, where the theory of the interruption of conduction at the edge of the excavation does not answer, when, as may happen, the sight is lost by degrees, accompanied by *sharply defined* defects in the peripheral field. V. Graefe has also here been able to watch with the ophthalmoscope the development of a choroido-retinal process, and to substantiate the proof of such a process by anatomical examination. But in reflecting upon the significance of these processes, he regards it as most probable that they also are the *results* of increased pressure.

If now we epitomize the views which v. Graefe supported to the last, we find that in his opinion all functional disturbance in glaucoma in the last line are inferred to be due to intraocular pressure, whether it be, that the conductibility of the fibres is interrupted at the edge of the excavation, or that an ischæmic retinitis is produced, or finally that there ensue disturbances of nutrition in the choroid and retina.

Rydel* has admitted that the hindrance to the flow of blood in the arteries of the retina has a decided influence upon the functional disturbance not only in acute inflammatory, but also in chronic and simple glaucoma, and has explained from the arrangement of the vessels, why it is that the periphery of the outer part of the retina first suffers in glaucoma, and consequently the visual field is first contracted inward.

But I believe that too great an influence upon the functional disturbance has been assigned to the intraocular pressure. The very preservation of vision in acute glaucoma ought to make us thoughtful. It is generally admitted that a suddenly increasing pressure is more destructive to the nerve substance or to the nutrition of the retina, than one increasing but gradually and slightly. This is true in-

* V. Graefe's Archiv, XVIII, 1, 1.

deed. But how then does it happen, when acute glaucoma with decided increase of pressure, occurs in a previously healthy eye, that the whole disturbance of vision is, as a rule, to be accounted for by the turbidity of the media, and that we have no contraction of the visual field?

And how, on the other hand, can we explain that in a directly contrary way in other cases, vision is wholly lost in the shortest time, without a notable increase in pressure? I have never yet seen a case of glaucoma fulminans, but v. Graefe† says explicitly that even in these cases where vision is lost irredeemably in even half an hour, the signs of increase in pressure need not be so very prominent in comparison with those in the usual cases of acute glaucoma. The connecting link between those cases in which a decided increase in pressure has prejudiced the nervous tissue neither directly nor indirectly by arterial ischæmia, and those in which the same pressure produces absolute blindness in the briefest period, is to be found in those cases of common acute glaucoma in which an early iridectomy fails to restore the sight. Although a condition resembling blindness, at least for the moment, can be produced by a decided pressure in every eye (new experiments have lately been made by Exner and Kühne on the causation of this peculiar pressure blindness), and although in a few cases of fulminating glaucoma the increased pressure may be so colossal that in it *alone* we find a sufficient explanation for the amaurosis; yet it is far from being proved that the increase of pressure, as is usual in acute glaucoma, takes any share in the functional disturbance, since the retinal function, with the same increase of pressure, remains in one case fully intact, while in another it is totally lost.

If we study further into the functional disturbances in excavation glaucoma, we must soon give up the idea that we have to do with the action of pressure upon the edge of the excavation. For similar causes must have at least somewhat similar effects, and besides we have already some knowledge of the functional disturbances, when in other affections of the conducting apparatus the layer of nerve-fibres is first attacked.

† Graefe's Archiv., VIII, 2,244.

If the amblyopia were due to pressure on the nerve fibres at the edge of the excavation, this sort of amblyopia ought to have something typical. In every case we ought to expect a sort of amblyopia such as we observe at other times when the percipient layer is intact and the conducting layer defective, and lastly, the amblyopia ought to correspond to the condition discovered by the ophthalmoscope. Accept, if you choose, a varying disposition for various individuals; in the two eyes of the same person you must expect disturbances of vision which correspond to the perfection and depth of the excavation.

The usual account of the state of the peripheral field of vision and of central vision in glaucoma is loaded down with so many exceptions that there can scarcely be a question of a rule. The diametrical oppositions are: at one time the central vision can be nearly or entirely lost without any perceptible defect in peripheral vision; at another time, on the contrary, the visual field shows extreme concentric limitation, while the central vision is wholly, or almost wholly, preserved. But we cannot procure such a diametrically opposite state of things from the appearance of the excavation, a fact that becomes especially striking when, with precisely similar excavations in both eyes of the same person, the vision of both eyes shows the greatest contrasts.

The right eye of Joseph Lindner gives us an example of lost central vision, with the field of vision preserved, while in the left eye of Joseph Rottmayer the field is everywhere extremely and almost concentrically contracted, but central *S* is almost, if not wholly, preserved, for the patient, æt. 66, has $S=\frac{20}{40}$. The two eyes of the previously-mentioned Lindner, æt. 61, offer a very interesting example, for while in *R. E.*, *F* is preserved, central *S* is lost; in *L. E.*, central $S=\frac{20}{30}$, but the peripheral field has undergone such an peripheral contraction (but not quite so extreme in all directions as in Rottmayer's case), that only the lateral-inferior quadrant is preserved, and even this is far from perfect. But from the appearance of the excavation, no one would have been able to discover this directly opposite state of the function of the peripheral and central parts of the retina.

As now there is no typical course of the peripheral and central amblyopia in glaucoma, and as there can be no question of a clue to the degree of one from the degree of the other, so on the whole there is no typical field of vision in glaucoma.

I have before me a full series of very careful field measurements (made by Dr. Plenk) partly from cases cited in this paper, partly from many others, and which represent a real chart of the anomalies of the field of vision. I will merely mention that it happens that not a single one of these pictures would correspond with that given, *e. g.*, by Schmidt,* as almost exactly typical. Instead of that, here *F* is free, there narrowed concentrically to a high degree; there it extends further outward than inward, there quadrants of the field preserved to the right and left, join at the point of fixation; and even where we can demonstrate so frequently a predominating limitation inward, the variations are still very numerous; here only the *lower* and outer quadrant, there only the *upper* and outer quadrant are preserved; here the line of demarcation generally runs obliquely from upward and inward to downward and outward, but in the horizontal meridian it extends far inward, there the defect is almost like that in hemiopia. Anna Pickelmayer, æt. 65, has in *LE*, $S=\frac{20}{30}$ almost, *F* free from defects; *R E* has the same central *S*, but there is a defect in the upper and inner quadrant, and in an adjoining sector of the upper and outer quadrant. I do not possess hundreds of carefully mapped-out-fields of vision, which would be necessary to statistics of any value; but this is superfluous for the following demonstration, which will show that this manifold variation in the functional disturbances with precisely similar appearance of the excavation, but especially the total independence of the central disturbance of the peripheral, and *vice versa*, cannot be explained by any pressure theory. If the functional disturbance must occur from direct pressure on the nerve fibres, then those most superficial ought to be affected first; if these lead to the periphery the periphery will suffer first, but on the other hand it will

* Graefe-Saemisch, V. I., 20.

suffer last if the fibres leading to the maculea lutea are the most superficial. But there ought to be here a certain regularity depending upon whether the arrangement of the nerve fibres is assumed in one way or the other. It is certainly asking a great deal to accept the assumption that, of the two eyes of the same individual, the central fibres lie uppermost in one eye, and the peripheral beneath, whereas in the other the peripheral fibres lie uppermost, and the central beneath; and yet according to the pressure theory, we must accept the existence of such a state of things, *e. g.*, in the eyes of our Joseph Lindner.

The theory of the nerve pressure accomplishes if possible, even less than the theory of the vascular pressure. Ischæmia may play an important *rôle* in a few cases of glaucoma fulminans (although for my part I am not convinced of that, in view of the fact stated previously, that even in glaucoma fulminans an unusual increase of pressure may be absent) but it would be forced to speak of it in the usual forms of acute glaucoma, in which the disturbance of vision is, as a rule, proportional to the cloudiness of the media, and in those in which this is not the case, there may be other causes than ischæmia (see case Deluggi); it seems to be strained argumentation, indeed, to ascribe an important *rôle* to ischæmia in chronic and simple glaucoma, as Rydel* has done. Leaving out of consideration the point that the manifold relations between peripheral and central amblyopia, and the varying forms of the peripheral defects of vision are opposed to the theory of our honored colleague of Krakan, yet we cannot, on the whole, talk of obstruction in the circulation in glaucoma simplex, for the increase of pressure is frequently but slight, and even problematical; even the symptom of occasional interruption of the arterial current (arterial pulsation) is as a rule not present, and the ophthalmoscope exhibits the usual distribution of the vessels in the periphery of the retina.

As, therefore, the above described functional disturbances in glaucoma, may, on the one hand, occur without excava-

* V. Graefe's Archiv., XVIII, I, 1.
† Klinische Monatsblätter, 1871, IX., 342.

tion, and, cannot on the other, be explained in a mechanical way, we are left to consider one other point, which up to this time, as it seems, has scarcely been looked at. That is to say we have but few comprehensive data on the state of the *sense of light* in glaucoma. I find nothing at all about it in Schmidt's monograph. Förster * speaks as follows: "I can not say anything satisfactory about glaucoma, because the results are extraordinarily different. We can really demonstrate a diminution of the capacity for perceiving light in those cases where glaucoma begins with inflammation, and in the prodromal stage; where it breaks out acutely S is so diminished that we cannot test it; in chronic cases I have found varying results; but it is certain that we do find cases after operations in which we cannot ascertain a diminution of the capacity for perceiving light." These words do not give us a deeper insight, but nevertheless they are worthy of consideration, by showing us that we already find diminution of L (light-sense) in the beginning of glaucoma. The significance which Förster attaches to the state of L is well known. When the parts *conducting* the sensation of light are especially affected by the morbid process, L is not altered, or only slightly. On the other hand we find great deficiency in L, "when the morbid process starts from the choroid, and affects at the same time the posterior layers of the retina, which *perceive* light."

What state then of L should we expect to find in glaucoma? Where we cannot speak of ischæmia, that is to say in chronic forms of glaucoma, the pressure theory assumes only an affection of the *conducting* elements, while the *percipient* layer, the layer of rods and cones, remains intact. Where we might grant that ischæmia came into play, it is not at all necessary that the percipient layer should suffer from insufficient nutrition, since owing to the circumstance that the outer retinal layers are absolutely bare of vessels, any nourishment of these layers from the choroid is at least as probable as from the retinal vessels. As I have already considered the physiological experiments on pressure blindness (although in all probability it has nothing at

* Zehend. Klin. Mon. Vol. IX., p. 342, 1871.

all in common with blindness from glaucoma, and rather imitates for us the state of embolism of the central artery of the retina), I will also mention that Kühne* does not agree with the view expressed by Exner,† in explanation of the appearances in these experiments, that we have to do simply with a disturbance of nutrition in the *receivers* of light, but rather refer it to the *conduction of light*, since "disturbances of the retinal circulation cause rapid blindness, even under circumstances where the retinal epithelium, the nutrition of which, in all probability, depends upon the choroidal circulation, is still intact, and only the ganglionic conducting apparatus affected." I only mention these details for the sake of accuracy. It is indeed, as Förster remarks, just in those cases of glaucoma in which ischæmia might perhaps come into play that it is impossible to test *L*, and on the other hand, in order to generally answer the question as to the seat of the disease in glaucoma, it will be needful first of all to study the state of *L* in glaucoma simplex. According to the prevalent pressure theory, since the seat of the disease is in the conducting layer of nerve fibres, we are not to expect a diminution of *L*, when the percipient layer is intact. But the opposite is the case in point of fact. In the beginning of the disease, when central and peripheral vision have still suffered but little, *L may* in spite of everything be intact, but in advanced glaucoma it seems always diminished, and usually in a high degree. Here we have no strict connection between the sense of light and the sense of space, and the state of things is the same as in retinitis pigmentosa, where too the degree of the diminution of *L* is not at all proportional to that of central acuteness of vision. Those cases are of great interest, in which, though finding the central and peripheral function still preserved, the increase of intraocular pressure, and the excavation doubtful, we can make an indubitable diagnosis by ascertaining a diminution of *L*. In the *R. E.* of Pichelmayer there were had the objective symptoms of glaucoma simplex; increase in pressure and total excava-

* Untersuchungen aus dem Physiol. Institute zu Heidelberg II., 1, p. 46, 1878.

† Pflüger's Archiv., XV., p. 409.

tion with the glaumatous ring; central S was still good; the upper-inner quadrant was absent, $L=\frac{1}{10}$; *L.E.* central $S=\frac{20}{30}$ almost, that is to say, so good that no one could affirm that it was pathological; F free, increase of pressure, and excavation but slightly pronounced, but here too L was $\frac{1}{10}$. So we might have diagnosed with certainty the beginning of the glaucomatous process in this eye. Still this diminution of L is only slight. To be sure it rarely reaches a high degree, even when central S is much diminished, as *e g.*, in Andreas Draxl's case where with just enough vision in his right eye only (fingers counted at 1′) to test him for L with Försters photometer, L was not less than $\frac{1}{18}$. Then again we see with well preserved central S an extreme diminution of L, as in the case of Lindner who has central $S=\frac{20}{30}$, defect of F in three quadrants, and L only $\frac{1}{60}$; in the case of Rottmayer, central $S=\frac{20}{30}$, extreme concricent narrowing of F, and $L=\frac{1}{81}$; and in the case of Auberger central $S=\frac{20}{60}$, upper and outer quadrants alone retained, L was even $\frac{1}{300}$. These valuations of diminished L, however, are really more significant than is expressed in the figures given, because the smallest necessary source of light regarded as sufficient for an eye with normal L to recognize the figures on the instrument, was 2 square *mm.*, while in our apparatus, and with our normal (astral) candle, a square, with a diagonal of 1½ *mm.*, *i. e.*, a square surface of $\frac{9}{8}$ *mm.* represented the smallest visible point of light for a normal eye, hence the last three valuations cited, $\frac{1}{60}$, $\frac{1}{81}$, $\frac{1}{300}$, correspond to $\frac{1}{9}$, $\frac{1}{144}$, $\frac{1}{533}$. After the subjective examination of Rottmayer's eye, we should have felt obliged to diagnose a typical retinitis pigmentosa, considering the preservation of central S, the extreme concentric contraction of F, and the great diminution of L, and yet the eye showed the most beautiful case of glaucoma simplex, with all the symptoms, and without any visible alteration in the choroid.

The state of L in glaucoma is in favor of the fact that the destructive process does not advance from the vitreous by pressure upon the layer of nerve fibres, but from the choroid by affecting the percipient layer, and it is

indeed noteworthy as bearing upon this point, that Förster found a diminution of *L* even in the prodromal stage of glaucoma, that is to say at a time when the functions are wholly intact between the transitory attacks. Although we shall later return to this subject, it is already clear that the moment we lay aside even for once the usual pressure theories, all the various forms of functional disturbance can be explained by the varying localization of this process in the choroid, and by its subsequent invasion, now here, now there upon the retina.

In the same way the state of the color-sense in glaucoma is in favor of the fact that the original cause of the amblyopia does not lie in a pressure-atrophy of the nerve fibres, for then we should recognize early disturbances in the capacity for perceiving colors. But according to my experience this is not the case.

The disturbances of vision may be far advanced, and yet the sense of color be perfectly preserved; and only when central vision has sunk very low, and atrophy of the optic nerve has become very pronounced, which in our view is secondary to the choroido-retinal process, do disturbances in color preception appear. When green blindness was found in the case of George Knabler, fourteen months after cure of glaucoma, with normal *T*, good central *S*, and intact *F*, without any trace of excavation, the approach of a genuine atrophy of the optic nerve was most to be feared, but there could be no question of a pressure atrophy.

Finally there are two conditions demonstrating that the functional disturbances are independent of pressure (leaving out of question the fact that the eye with the less pronounced excavation often exhibits the greater disturbance of *S*); one that after normalizing the pressure, *S* often declines abruptly, the other, that glaucoma can be cured, and as it seems, permanently cured by iridectomy, although the eyes on which it has been performed show such a degree of *T*, that if one did not see where the iris had been excised, and were not informed of the integrity of the function, one would recommend the immediate performance of an operation for glaucoma. We have described cases of

the first kind in detail, at a previous page. In point of fact, if the functional disturbances were due to pressure on the optic nerve, it would be far beyond our faculty of comprehension (of which, by the way, in the doctrine of glaucoma too much is demanded), that the act of freeing the optic nerve from this destructive pressure should be followed immediately by a rapid deterioration of its function.

Cases of the opposite sort are to be classed with acute glaucoma. Then we see eyes which continue perfectly cured, although iridectomised years before, but which are so hard to the touch that we are astonished. I have also seen eyes that had been operated on ten and more years before for relapses of inflammation in spite of permanent blindness, and since then were perfectly cured, and which, however, with very moderately developed excavation, were as hard as bone.

If we review now all that has been said in this section, we come to the irrefutable conclusion, that the functional disturbance in glaucoma is not caused in a mechanical way, not by pressure on the optic nerve and retina, but also, that in glaucoma simplex we cannot assume, as v. Jäger has done, a simple, primary affection of the optic nerve, since then the functional disturbance could scarcely manifest itself otherwise than in a pressure affection of the optic nerve.

III. Inflammatory and other symptoms.

Schnabel* has lately denied an inflammatory character to the irritative symptoms in glaucoma. He says that the corneal cloudiness is produced by the secretion of a cloudy fluid, and is to be regarded as the result of a neurosis of secretion. Iridectomy removes at one sweep the cloudiness of the cornea, the pain and the increase of pressure.

The iris, although occasionally very rigid, is not inflamed, for the pupil in glaucoma is wide, free from synechiæ, responsive to atropia, never blocked up by exudations, and the tissue of the iris reacts to iridectomy as in the healthy condition, while the same operation when performed on an

* These Archives, Vol. V., page 351.

inflamed eye adds to the inflammation, and may even lead to phthisis. Pain also and episcleral injection are the expression of a neurosis, while the cloudiness of the aqueous and the vitreous does not need to be explained, because it does not exist. Glaucoma excludes the cloudiness of the vitreous, and Schnabel has never been able to convince himself directly of any cloudiness of the aqueous.

As to the history of the cloudiness in glaucoma, I should like to remark that Schweigger did not, as Schnabel says, first call attention to cloudiness of the cornea, and declare his doubts of turbidity of the aqueous, in the third edition of his handbook (1875), but that he had already done this in the first edition (in 1871, page 501), and that later he only added that he had never been able to satisfy himself of the coexistence of a diffuse turbidity of the vitreous. But to be exact, it must be added that Liebreich* had long before recognized and accurately described the cloudiness of the cornea in glaucoma. Liebreich says that with help of the microscope, he had already recognized years before that the periodic cloudiness in glaucoma does not have its seat in the aqueous, but in the cornea, and particularly in the epithelium and most anterior layers. I desire here to say, as regards Schnabel's view, that the moment the cloudiness becomes objectively pronounced in any noticeable degree, there could be no question of confounding the cloudiness of the cornea with that of the vitreous, since any cloudiness that veils the iris cannot be located in the vitreous, but only in the aqueous or cornea.

For myself I find no reason for regarding the visible alterations in the cornea and iris as non-inflammatory, although I would not wholly deny the cloudiness of the aqueous and vitreous. Schnabel himself has experienced how one may be decived, for later when examining the aqueous in cases of inflammatory glaucoma, he found it really cloudy—by pigment molecules, not by pus globules, but still cloudy. In the same way there might be a cloudiness of the vitreous, and this would not at all militate against the theory of neurosis, because if a neurosis of secretion

* Klinische Monatsblätter, 1863, pag. 488.

can deposit a cloudy fluid in the cornea, I see no reason why the same mechanism should not be able to deposit a similar cloudy secretion in the aqueous and vitreous, from which, as I believe, they would be more sure to disappear quickly than from the fibrous tissue of the cornea.

I will also suggest that whoever admits the inflammatory nature of the cloudiness in the media, and of the process in the iris in glaucoma, need not for this reason cease to be a neuro-pathologist in the province of glaucoma, for it is the ophthalmologist especially who has an undeniable example of the origin of the most undoubted, most severe inflammation by direct nervous influence in sympathetic cyklitis. Just as a plastic cyklitis is produced by irritation of the nerves in sympathetic ophthalmia, so might another form of inflammation of a more serous character be produced by irritation of the nerves in glaucoma.

But inflammatory processes in glaucoma are undeniable. We are no more authorized in claiming the intervention of the nerves for these processes than in regarding every other form of keratitis or iritis as the expression of neurosis.

The cornea shows the signs of an interstitial keratitis. In its various gradations of cloudiness from the slight haziness of the membrane to the most intense opacity and in its course, which never leads to suppuration, but either to a perfect clearing up, or to parenchymatous and permanent opacities, the keratitis in glaucoma coincides with the usual form of non-vascular interstitial keratitis with which it may have also a relationship as regards its long duration, since the keratitis in glaucoma may continue for months, and even for years, and finally disappear of its own accord, either wholly, or leaving deep spots in the cornea, which are often found after a close examination in old cases of glaucoma with otherwise transparent corneæ. This keratitis in acute glaucoma, where the cloudiness is marked, is usually arranged partially in stripes, which on lateral illumination show an intense grayish, and even decided yellow tint (very much like the stripes that we see developed so often after v. Graefe's cataract operation), run in various

directions, and may be confined to the very centre of the cornea, while the periphery shows a diffuse infiltration. A description of this cloudiness has been given already in a special case. This keratitis yields rather rapidly to the operative procedures for the glaucomatous inflammation.

But it is not right to say that it disappears immediately after the operation, for that would conflict with the meaning of the cloudiness as an inflammation. Schweigger* indeed says that this cloudiness disappears after the operation, but "not at once, as many report, but usually not until from eight to fourteen days." Its course even to complete disappearance has been depicted in a case before described, but perhaps this case is not a type. I had never seen the stripes change into squares before, but I was repeatedly able to prove that it lasted for twelve days, till the cornea became quite clear, after an iridectomy for acute glaucoma. I have also seen a case where it took several weeks before the keratitis began to improve after an iridectomy, the only case in which I saw keratitis constitute the only objective symptom of glaucoma. Baroness E. of South Tyrol had lost the sight of the left eye some time before with the symptoms of acute glaucoma, with the usual course. When seen for the first time the globe was hard, media clear, excavation, amaurosis. The patient said that she had lost the sight of her right eye within a few days in the same way, with the same pain, and to about the same degree as previously in the left eye. The symptoms are, marked ciliary injection, slight chemosis of the conjunctiva, thick, central, grayish, parenchymatous cloudiness of the cornea sufficient to explain the disturbance of sight (fingers counted, *F* free), but no increase of intraocular pressure, no shallowness of the anterior chamber, nor widening of the pupil, which reacted sluggishly. Up to the time that I saw this case, I had not generally turned my attention to cloudiness of the cornea in glaucoma. It seemed quite strange to me that a simple keratitis should fully explain all the appearances of acute glaucoma (pain and disturbance of function), and that the process was, however, acute glaucoma, was

* Volkmann's Vortralge, No. 124, pag. 1035.

proved by the history, and by observation of the second eye. But now the matter is simple. Keratitis may sometimes appear as the very first of the inflammatory symptoms that constitute glaucoma, since it, as well as the functional disturbances, is independent of increased pressure; other glaucomatous symptoms will undoubtedly follow it. The iridectomy performed for this primary glaucomatous keratitis removed the pain, as usual, but the cloudiness of the cornea did not yield for several weeks, and hence vision was not improved. At last the keratitis disappeared, restitution followed, and a permanent cure.

It is a fact too evident and too generally known to admit of doubt, that the iris is inflamed in an attack of acute glaucoma, from the fact universally recognized that the bit of iris excised appears rigid, firm, infiltrated and thickened, as also that a single attack of acute glaucoma sets up alterations in the iris, which either do not disappear at all, or in the most fortunate cases disappear only after the lapse of some weeks. The iris must become rigid from some cause; its tissue must be altered permanently or temporarily by some cause. Microscopic investigations seem to recognize this characteristic in all glaucomatous inflammations, that there is but a slight representation of newly formed elements in the products of inflammation; but that extreme alterations also are produced by a sero-fibrinous exudation (which we shall therefore be obliged to assume), we see from the alterations of tissue in the iris, and from the fact that the sphincter may become permanently paralyzed, not perhaps by pressure upon its nerves, but as a result of inflammatory infiltration in acute glaucoma, as is convincingly proved in those cases healed by sclerotomy, in which the operator's hand has spared the sphincter. Where pressure on the nerves was the cause of the stiffness of the pupil, its mobility returns after sclerotomy.

But leaving aside all that has just been said, I desire to remark as regards what Schnabel says, that the iris in acute glaucoma, before, during and after the operation, does not behave at all normally. A drop of eserine, that would before have contracted the pupil completely, does not act in

acute glaucoma, and it is only after repeated application of the drug that we can obtain a certain action; likewise, in my opinion, it is not proved that such an eye behaves differently towards atropia than towards eserine. Synechiæ are indeed usually absent, but not always. The formation of synechiæ and pseudo-membranes in the pupil is almost impossible, owing to the character of the inflammation and the dilatation of the pupil by pressure upon the nerves of the iris. Is the usual iritis serosa no inflammation, because it shows no synechiæ and no exudations in the pupil? Moreover, pressure in the ciliary region causes as much sensitiveness in acute glaucoma as in acute iritis.

During the operation the iris behaves as in an undeniable inflammation, since the seizing, the pulling out and the excision are painful, and often so in a high degree. And no one can assert that all reaction is absent after the operation; for in spite of the favorable influence on the whole process, it is not rare for us to find that the increase of the inflammation of the iris leads to synechiæ. And when Schnabel cites the fact, "that we sometimes find synechiæ between a few points of the anterior surface of the iris and the cornea, after iridectomies in inflamed eyes," I may say that I have seen these true anterior synechiæ twice without perforation of the cornea, but noticeably both times after iridectomies for glaucoma, and still further, both times without violent inflammatory symptoms. In the first case, however, with previously existing posterior synechiæ, I could not confirm the fact that the iridectomy in inflamed eyes always causes inflammation, for I often perform iridectomy for chronic iritis, and of all the good results of iridectomy, I know hardly another that can equal it in brilliancy and constancy in such cases. However, when irido-cyklitis is present, phthisis frequently follows, although the most violent pain disappears instantaneously. But when a plastic iritis has frequent relapses, or kindles anew and anew without any real intermission; when an iritis serosa lasts for months and even years, and drives both physician and patient to despair then there is but one cure—iridectomy. Objective symptoms of irritation increase, synechiæ form or enlarge,

directly after the operation, just as in acute glaucoma, but after this traumatic irritation has passed away in a proportionally short time, we obtain a recovery, and as I have convinced myself in those cases that I have observed for years, a lasting recovery.

A kerato-iritis, therefore, that begins with violent ciliary injection is a partial symptom of acute inflammatory glaucoma. The cloudiness of the pupil is of course usually produced by the cloudiness of the cornea, or rather we are as a rule unable to prove that there is also a cloudiness of the aqueous, for the common observation that the pupil and iris are *a trifle clearer* after the aqueous has escaped, is to be accepted with reserve, in so far as we consider that the optical relations are altered by the emptying of the anterior chamber. But there are cases where the cloudiness in front of the iris is so dense that we can scarcely see the iris or pupil, while, when the aqueous has escaped, both iris and pupil are at once distinctly seen. Unfortunately, I have not seen any such cases since the question of the turbidity of the aqueous has become a matter of dispute; when I saw them, I wondered at the thickness of the turbidity, but not at all at its disappearance after the puncture. I regard it as incredible that a cloudiness of the cornea of such density as to make the iris and pupil quite invisible, should at once disappear after puncture of the cornea. Nor could the cloudiness of which I am now speaking be due to pigment molecules of the iris, because the color is a uniform gray, and I have seen it disappear quite rapidly in those cases in which they finally were removed at once and forever by an iridectomy performed during an attack.

I do not believe that pigment molecules in such masses can be absorbed so rapidly. But every one who has seen how a considerable hypopium in traumatic keratitis disappears in a night after the employment of atropia and the pressure bandage, will know with what exceeding rapidity the products of inflammation will disappear from the eye under certain circumstances.

I have nothing decisive to say about diffuse cloudiness of the vitreous, for since the question has been in dispute, I

have seen no cases in which turbidity of the media could be diagnosed objectively while the cornea was still clear. On the other hand, I cannot refrain from saying that in a few cases of the purest forms of acute glaucoma, in which the ophthalmoscope showed the choroid to be intact, I watched the development of scattered, dense opacities in the vitreous, close to the retina.

I have this to say about the turbidity of the media: so long as it is slight, and the function normal in the interval of the attack, the glaucoma is, according to Graefe, in its prodromal stage. But there is frequently only a gradual distinction between these observations and the acute attack of glaucoma, for even after such an attack the function may return spontaneously to its normal state; and I have seen cases in the condition of the prodromal stage (so also of glaucoma simplex with almost intact function) in which the patients declared that they had passed through attacks of blindness which, in twenty-four or forty-eight hours, again gave place to the former vision. But how long can these attacks continue until the final and permanent signs of glaucoma appear, and we can no longer speak of a prodromal stage? Prodromal stages of many years' duration have been described; but this stage may last a full half-century, as I learned from the case of Mr. S., whose right eye I iridectomized at Innsbruck in 1874. Mr. S., 70 years old at the time of the operation, declared, with the greatest positiveness, that he had had attacks when quite a young man. When he frequented the beer-houses during his student life he used to have frequent circumorbital pain and headache, a cloud before his eyes and colored flames around the lamp lights. All his attempts to rub the clouds away from his eyes were fruitless. He had to stop smoking at once, and when he did this, the next morning his sight was clear again. These attacks were repeated during the space of fifty years, but he had long since discovered the only remedy to relieve them—sleep. As the attack came on at the most varying parts of the day, the patient has also learned to go to sleep at any hour, an art which, in his case, did not need a long experience to gain, since so many glaucomatous pa-

tients extol this well-proved remedy. I made an iridectomy at last to remove the attack, and also because the vision had begun to suffer, while the excavation was becoming more and more distinct. This case, by the way, was marked by most intense turbidity of the aqueous, which disappeared at once and forever after the puncture.

If we consider merely the fifty years duration of these attacks, our only alternative is to choose between the greater probability of a neurosis lasting for half a century, or an ever-recurrent inflammatory cloudiness of the media, particularly of the aqueous, lasting for half a century. Nor do I know whether the circumstance that such cases sometimes yield only to sleep, and do not disappear until sleep ensues, is in favor of a neurosis or of a disturbance of circulation increased to an inflammatory cloudiness. We have a brilliant example of the latter sort in a young man with a high degree of hypermetropia (October 5th, 1877), who has suffered for a year and a half from periodic pains in the left eye, coming on at evening, and beginning with halos and which at one time had increased to a very violent inflammation, with extreme diminution of sight that had lasted for forty-eight hours. For the last few weeks the attack has come on every evening at 9:30 o'clock; and here let it be said, that a course of inunction, lately resorted to, had no influence, while by the use of quinine (or after it) the pain disappeared, and the halos alone remained, so that at present he sees rainbow colors every night at 9:30 o'clock. The examination by reflected light during the attack shows no turbidity of the media, but by the use of direct light we could convince ourselves of a slight corneal cloudiness, which however, in my opinion, did not suffice to explain the intensity of the rainbow colors. Since these evening attacks first appeared, they never disappear so long as sleep does not intervene. If the patient passes the whole night at a ball or elsewhere, the new day does not free him from halos. Sleep alone does this.

Having mentioned the excavation, the functional disturbance and the inflammatory phenomena, we have still to describe the symptoms in the anterior section of the

globe, known as phenomena due to pressure, before we can draw from all our observations a conclusion as to the nature of the glaucomatous process. Development of the ciliary veins around the cornea, anæsthesia of the cornea, shallowness of the anterior chamber, widening and immobility of the pupil, may or may not be symptoms of pressure (the pupillary symptoms which disappear after sclerotomy are caused by pressure); but all these, like arterial pulsation, have not the slightest significance as regards the glaucomatous process, since they all are absent in true glaucoma simplex. Here, with complete blindness and deep excavation, the tension may be very high, the globe indeed hard—we may, perhaps, discover one or two sub-conjunctival venous trunks, but the cornea is perfectly transparent and sensitive (if we act very rigorously in such cases, and believe that we ought to regard the sensibility as lessened, let us not forget that the sensibility of the cornea in normal circumstances varies a great deal in different individuals), the anterior chamber not at all shallow, the iris normal, the pupil clear, and even in the amaurotic eye (only consensually, of course,) reacting most promptly. We saw prototypes of true glaucoma simplex in the left eye of Kapilles and in the right eye of Pottmayer. We have only to call attention to the fact, that the increase of pressure in inflammatory glaucoma ought to lead to the well-known pressure symptoms on the part of the ciliary nerves, while, as v. Graefe says, the idea is inadmissible "that the increase of pressure hinders the nervous conduction as such," and "that the nerve substance in general tolerates a very considerable pressure."* He says this of the nerve substance *in general*, and hence, in his opinion, although he refers only to the optic nerve and retina, the assumption is inadmissible, that anæsthesia of the cornea, dilation and immobility of the pupil, are caused by pressure-paralysis of the ciliary nerves.

As v. Graefe also says, "A much greater degree of pressure is needed to disturb the conductibility of the optic nerve fibres, and of the middle and outer retinal layers, than that which is active in glaucomatous processes." * * *

* V. Graefe's Archiv., XV, 3, 112.

Hence the assumption of ischæmic paralysis of the retina in acute glaucoma. But I say: if the ciliary nerves yield to a pressure to which the substance of the optic nerve does not yield, then the latter will surely not yield to a pressure to which even the ciliary nerves do not yield; *i. e.*, if the pressure in glaucoma simplex cannot at all affect the ciliary nerves, so much the less will it be able to affect the optic nerve and the retina, and hence there is no foundation whatever for the view that the functional disturbance, as well as the excavation in glaucoma simplex, are the sequences of increased pressure.

The symptom of increased intraocular pressure loses its significance in glaucoma, since in this disease (with the exception perhaps of a *few* cases of glaucoma fulminans), we deny to it any sort of an initiative in the introduction of the functional disturbance. But we should not, as Schnabel has done, use the fact of increase* of pressure to militate against the view of glaucoma as an inflammation, for after having directed my attention to this subject for years, I have come to acknowledge the theory which I have already expressed elsewhere,† "Increase of pressure may show itself in every acute inflammation of the eye, no matter what its nature or what its causation." But scarcely any one need assent to Schnabel's statement "that manifest inflammations on the whole, rarely if ever call forth increase in pressure," leaving out of consideration that I found increase of pressure in every keratitis, plastic iritis, choroiditis and even cyclitis, because there is a manifest inflammation, iritis serosa, which begins almost constantly with an increase of pressure, which may be of long continuance. The increase of pressure in iritis serosa shows us one instructive fact, that it may last for weeks, months, a year, and even more, without prejudice to the function of the retina and optic nerve. It proves to us that an increased pressure which really acts only from the vitreous chamber outward upon the retina, does the retina no harm. The iritis serosa yields at last, and with it the increase of tension

* These Archives. Vol. V, 367.
† Die Sympathischen Augenleiden, pag. 56. 1878.

without any harm to the retina and optic nerve. Only when the process has passed over from the iris to the choroid do functional disturbances and excavation of the optic nerve develop themselves. We say, then, that secondary glaucoma has supervened. Just as the increase of pressure may be absent in glaucoma, so, on the other hand, it may last for a long time without giving rise to glaucoma as the example of iritis serosa shows. Indeed, I am of the opinion that many processes beginning with a great increase of pressure, and the usual accompanying symptoms that are observed on the part of the cornea and iris, are very different from glaucoma. I desire in this class to reckon the glaucoma hæmorrhagicum, which, in its beginning, its course, the nature of its functional disturbances, and in its results, has no similarity with typical glaucoma. It begins with hemorrhages in the retina, does not lead to excavation, destroys vision by the hemorrhages in the retina, and is but slightly influenced by iridectomy. Especially would I point out, amongst other things, that the destruction of vision does not ensue as in glaucoma, and that in no way can we expect its restitution. For, as the previously cited ophthalmoscopic examination shows, vision is lost by hemorrhages in the retina. Galezowski's case * shows in this connection a still more interesting ophthalmoscopic picture ; five months after the performance of an iridectomy in a case of hæmorrhagic glaucoma, he found "a total absence of the vessels on the papilla."

Since we rest the diagnosis of glaucoma, not upon the recognition of a decided morbid process, but upon the presence of certain symptoms, the question presents itself, whether, amongst these symptoms, we must have a pathological increase of tension in order to diagnose glaucoma. This formerly seemed to me justifiable, when the nature of glaucoma was not recognizable. Since the excavation of the optic nerve is not constantly present, we should therefore define glaucoma as that disease which, accompanied by constant, although perhaps varying increase of pressure, leads sooner or later to lasting disturbances of function, and finally (if not always) to blindness, so that that disease in which the

* Journal d' Ophthalmologie, 1872. Receuil d' Ophthalmologie, II, 77.

excavation of the optic nerve and blindness are developed without increase of pressure, should not be called glaucoma, but "a glaucomatous affection of the optic nerve."

At any rate, it would be more scientific, if we tried to distinguish by the term glaucoma not a definite group of symptoms, but a definite process. Then, too, excavation of the optic nerve without increase of pressure, would find its proper place, while, on the other hand, we should exclude those processes beginning with continuous increase of pressure, and leading to blindness, but which, by their very nature, are decidedly different from glaucoma.

IV. Nature and Theories of Glaucoma.

From the sum total of facts presented, it is not difficult to draw the conclusion at which I must have arrived, concerning the nature of the glaucomatous process. Glaucoma is a choroiditis, complicated also in the anterior segment of the globe, with inflammatory symptoms, which have their origin in various regions of the choroid, and may thus affect the retina at various points, and which, as a rule, begins with increase of tension, but also progresses without this, and shows its individuality not only by the increase of tension that usually accompanies it, but also by the development of an affection of the optic nerve, which is dependent upon the process in the choroid, but is not always present. On the one hand, the process may be insidious throughout (glaucoma simplex, with or without increase of tension), or, in spite of its insidious nature, there may be now and then noticed the appearance of distinct inflammatory symptoms (turbidity of the media, keratitis), which latter, however, do not lead to permanent alterations of structure in the anterior segment of the globe (glaucoma simplex, with intercurrent and visible phenomena of inflammation).

On the other hand, the inflammatory symptoms of the anterior segment of the globe may develop from the outset suddenly with the greatest violence, while the process in the choroid does not suddenly rise to an an analogous severity, and hence there is an absence of a sudden and decided reaction upon the retinal function (acute inflammatory glaucoma), or

again this choroidal process bursts out with the same intensity and in similar character, as in the anterior segment of the globe, and injures greatly the function of the percipient layer of the retina, or even utterly destroys it (acute inflammatory glaucoma with abnormal course, and fulminating inflammatory glaucoma).

Furthermore, there are cases in which the choroiditis alone is markedly developed either without or with but insignificant inflammatory symptoms in the anterior segment of the globe, and so leads rapidly to impairment or destruction of vision (glaucoma acutum, resp. fulminans, without visible inflammatory symptoms). It is by no means seldom that the history of the case shows that the blindness ensued during the night without pain, or with but slight feelings of pain, so that only when the patients awoke did they become aware of the extreme defect of their vision. These are cases of the ordinary *acute* glaucoma, as the further state of vision shows. But v. Graefe * has already observed that even in glaucoma *fulminans* vision may be totally lost without pain. Finally, the inflammatory symptoms in the anterior segment of the globe, as well as the choroidal process, may assume a chronic course (glaucoma inflammatorium chronicum).

The functional disturbance in glaucoma cannot be explained by a primary affection of the light-conducting apparatus; it depends upon some injury done by the choroiditis to the function of the light-perceiving apparatus. Increase of tension and glaucomatous affection of the optic nerve, both of them accompanying symptoms of choroiditis, can only hasten the end; but they are neither the cause nor the beginning of blindness. This ensues, as we know by direct observation, even without increase of pressure, in pronounced affections of the optic nerve, or with increase of pressure without excavation of the optic nerve. But in consequence of this, it is quite clear that there must also be real glaucomatous processes which lead to blindness, *without* increase of pressure, and *without* excavation, so that the mirror at the time reveals no alteration, at least in the easily accessible parts of the fundus, or merely the secondary, simple atrophy

* V. Graefe's Archives, VIII, 2, page 250.

of the optic nerve. We shall only be able to diagnose glaucoma in these cases, when we shall have learned to rest our diagnosis of glaucoma not upon the formation of one symptom, the increase of pressure or the excavation, but on the knowledge of the process in the choroid, that lies at the bottom.

In considering the nature of this choroidal process, there is no doubt that its products, like those of glaucomatous keratitis and iritis, are poor in new-formed elements, and when Hock is of the opinion* that Schnabel's view of glaucoma is not satisfactory, but that we have to thank him for one thing which cannot be esteemed too highly, namely, that he has entirely done away with the choroiditis serosa, I cannot by any means agree with him. The choroiditis which occasions the glaucomatous process, and also affects the vascular region of the scleral circle (v. Jäger), cannot be remotely connected with choroiditis serosa.

"It would be impossible to convince ourselves," says Schnabel,† "that an inflammatory process lies at the root of glaucoma simplex," since "we cannot recognize in it a single undeniable inflammatory symptom." I would remark that, if even up to this time, not a single undeniable inflammatory symptom had been recognized in glaucoma simplex, this could not be cited as a strict proof against the non-inflammatory character of this affection. Schnabel,‡ who so felicitously opposes the generally accepted inflammatory nature of certain symptoms, contradicts himself by declaring as an inflammatory product a symptom of glaucoma *simplex*, which, up to this time at least, has never been thought to be inflammatory, namely the scleral ring which, as Schnabel says, is but exceptionally absent in glaucoma simplex, and is not the effect of pressure, but constitutes an independent "disease" of the lamina cribrosa, and of the choroidal region surrounding the optic nerve. Hock§ is right in asking what sort of a disease that could be if not inflammatory? Schnabel has later learned that even in glaucoma simplex, there may be extensive

* Wiener Klinik, 1878, pag. 179. † These Archives, Vol. V, page 343.
‡ These Archives, Vol. V, page 370. § l. c., 178.

products of inflammation in parts of the eye which are inaccessible to observation. He found, after making an iridectomy in the eyes of a person affected with glaucoma simplex, a pathological membrane, and in the right eye even a vascular membrane in the posterior chamber, referable to the posterior surface of the iris, and makes a report of similar discoveries by v. Jäger and Kerzendorfer.* Since, moreover, Schnabel expresses the view that this pathological formation, which can not have arisen from a neurosis, stands perhaps in important connection with the glaucomatous process, we, looking at the other views expressed in his last paper on glaucoma, oppose, perhaps unnecessarily, his theory of neurosis, since possibly he no longer clings to it.

I would, however, by no means admit that it will not be possible, even without the support of new means of diagnosis, to recognize during life the choroidal process that constitutes glaucoma. If the accounts given by Fuchs† prove to be correct, the process appears in the most anterior segment of the globe, as atrophic choroiditis. At all events it is very different from the usual form of exudative and atrophic choroiditis, for the presence of increased pressure and excavation of the optic nerve in exudative choroiditis (see the case of Alois Mayer,) is one of the greatest rarities (even v. Graefe saw only two cases of the sort‡), so that from this very reason it seems quite incomprehensible to me how any one can regard increase of pressure (glaucoma) as the *result* of choroidal atrophy. The choroiditis, which is glaucoma, does not at first lead to remarkable alterations, at least not in the fundus of the eye; but I should like to direct attention to the pigment epithelium, which, on careful examination in the erect image, shows, amongst other things, some delicate alterations. Slight as these alterations look ophthalmoscopically, the process which lies at their foundation is threatening to destroy the layer of rods and cones.

The atrophy of the choroid, which is the result of the glaucomatous process, has often enough been proven to exist

* These Archives, Vol. VII.
† Hirschberg's Centralblatt. Beilage zum Augusthefte, pag. 15.
‡ V. Graefe's Archiv., XV, 3, 173.

by the microscope. So, too, the atrophy of the ciliary muscle, which Brailey and Schnabel describe as an almost constant change even in relatively fresh cases of glaucoma, is evidently not to be regarded as the cause, but as the result of the glaucomatous process. It often seems to lead above all to atrophy of the ciliary muscle, although this does not involve the eye in any further damage. The functional disturbance, and indeed also the increase of pressure, ensue when it has extended to the posterior parts of the choroid.

I refrain from any remarks on the curative action of the glaucoma operation, and also on the causes of the glaucomatous process, nor will I speak of the theory of "impeded outlet," which has been refuted by Schnabel and others. I will only remark that the process is the same in the so-called secondary glaucoma as in the primary, and that a wound sometimes plays a part amongst the causes of pure secondary glaucoma. I cite this with reference to the words of v. Graefe:* "Glaucoma could scarcely be caused in a direct way by a wound in a perfectly healthy eye, and hence an intimate etiological connection between a wound and glaucoma could not be assumed, a result that perhaps is not wholly irrelevant for the comprehension of glaucoma."

Johann Steinlechner, æt. 45, presented himself January 20, 1870. He had received a violent blow on the left eye a short time before. A severe and unusually painful inflammation followed, and vision remained poor after it at last had passed away. *R. E.* is perfectly normal. *L. E.*, pupil somewhat widened, and reacting sluggishly. Ophthalmoscope showed anterior cortical cataract, but no trace of luxation of the lens, and deep glaucomatous excavation. *T* distinctly increased. *S* barely $\frac{1}{10}$. *F* contracted.

* V. Graefe's Archiv., XV, 3, 221.

SARCOMA OF THE TARSUS AND CONJUNCTIVA OF THE LID, WITH AMYLOID INFILTRATION.

By J. S. PROUT, M. D., of Brooklyn, N. Y.,

AND

CHARLES S. BULL, M. D., of New York.

I. CLINICAL HISTORY AND OPERATION.

By J. S. PROUT, M. D.

HULDA F., æt. 18, German, consulted me October 9th, 1878. Some years ago she was treated by Dr. H. Knapp, of N. Y. City, for granular lids. About four weeks before coming to me, she noticed a small swelling, like a stye, in the lower lid of the right eye near the outer commissure, which had grown rapidly, but without giving any pain. She could assign no cause for it. On inspection the conjunctiva of the lids of both eyes was found to be trachomatous to a slight degree. Occupying the conjunctiva and tarsus of the outer half of the lower lid was a nearly smooth, flat growth, with a surface slightly concave, of dense tissue, which extended from the tarsal margin downward beyond the cul-de-sac, and outward a little beyond the commissure. The skin was not at all involved but slid freely over the outer convex surface. The lid was not painful when handled, and could be easily everted, when the whole inner surface of the tumor came into view, presenting distinct rounded outlines, which, at its inner margin, rose about two millimeters above the conjunctival surface of the rest of the lid. It measured about 24 *mm.* transversely, 16 *mm.* vertically, and 2½ or 3 *mm.* in thickness at the edge of the lid. In color it was a light translucent red, and suggested a large, dense, chronic, trachoma mass. There was no active inflammatory condition of the neighboring tissues, but, as before remarked, a slight granular conjunctivitis; no glands of the face or neck were enlarged. The cornea was not at all affected, nor was there any impairment of vision.

I could not make a diagnosis. I saw no reason to suppose it syphilitic, or a wart, or an epithelioma, or, as the tarsus contains no true cartilage cells, an enchondroma.

As there were no urgent symptoms, calomel was dusted over its surface, a mercurial ointment was given for external application, and a borax and camphor wash prescribed. As no change for better or worse followed, I sent her, on October 22nd, to Dr. H. Knapp with a request for a diagnosis, and suggestions as to treatment. He was uncertain as to its character; said he had seen somewhat similar trachoma masses, and suggested a trial of the sulphate of copper. During the next six weeks this was applied eighteen times without effect on its size, but it became sensitive. I therefore (on December 9th) presented the patient at the meeting of the Ophthalmological Society. No member present had ever seen anything like it; there was no agreement as to its character, but all agreed that it should be removed with the least possible destruction of tissue. On December 28th she complained that for a few days the growth had been very painful, the pain extending down on the cheek, and she wished to have it removed. I therefore operated on the 31st at the Brooklyn Eye and Ear Hospital, Dr. Rushmore administering ether, Drs. Mathewson and Sizer assisting, and Drs. Bull and Hinton, of New York, present.

An incision was carried along the margin of the outer half of the lid, following the mouths of the Meibomian follicles; the lid was then everted, and the conjunctiva and tarsus being divided close to the inner (nasal) side of the tumor, this was, with very little difficulty, and but little bleeding, dissected out. It was found to have no special adhesion; did not involve the orbicularis muscle, and extended below the lower cul-de-sac, carrying the conjunctiva with it. In order to expedite healing, the cut edges of the lid and conjunctiva were stitched together, it not being thought advisable then to endeavor to prevent a symblepharon, but rather to operate on this afterwards if found necessary, when healing and contraction were completed. Union took place promptly, and now (March 4th, 1879) there is about one-third of an inch of conjunctiva between cornea and edge of lid, the latter being slightly drawn in, with very slight deformity or interference with the movements of the ball.

Feb. 19th, she complained that for several days past there had been severe bilateral circumorbital pains, which came on at eight

o'clock in the evening and lasted an hour with no accompanying ocular injection nor dimness of vision, for which no cause could be found. I directed her to take 5 minims of the fluid extract of Gelsemium semp., (made by Dr. Squibb, representing its own weight of the root), at 5, 6, 7 and 8 P.M. She took three doses, which produced the physiological effects of the drug; the fourth caused nausea and vomiting, but the pain was relieved. For the next four days she took 5 minims at 6 P.M. On March 4th she reported that the pain was entirely relieved.

The growth when removed was flat, irregularly oval in shape, with rounded distinct edges, slightly convex exteriorly, concave on its ocular surface, which was white and glistening. There was no ulceration. The tissue was brittle, translucent and only slightly vascular. It measured 24 *mm.* in its long diameter, about 17 *mm.* in breadth from before backward or above downward, and 4 *mm.* in thickness. On cross section when fresh, there appeared what looked like a faint outline of the tarsus.

Consulting Wecker's "Mal. des Yeux" and "Thérapeutique Oculaire," Graefe-Saemisch's "Handbuch," etc. etc., nothing that can throw any light on the nature of the neoplasm has been found, except in Wecker's "Thérapeutique Oculaire," page 128, where he says, that amyloid degeneration of the conjunctiva causes the highest degree of hypertrophy of the conjunctiva that one ever finds. It involves especially the upper half of the cul-de-sac and the semi-lunar fold, which become so much thickened that they protrude through the lids, presenting yellow gelatinous masses which suggest granulations. Judging from the small number of cases which have yet been observed, this disease is differentiated from granulations by its restriction to a limited portion of the conjunctiva, the neighboring parts not being involved. "En outre, il paraît qu'il s' opère, par suite de la dégénérescence amyloïde des parois vasculaires, des épanchements sanguins étendus que l'on ne rencontre guère dans les cas où il s'agit d'un dépôt en masse de granulations." This condition did not exist in my case. He recommends removal of the affected tissues.

II. MICROSCOPICAL EXAMINATION.

By CHARLES S. BULL, M. D.

THE growth had an irregular quadrilateral shape, with the corners more or less rounded, and the edges of the upper surface bevelled towards its centre. This made the upper surface of a generally concave aspect, the concavity being deepest at the centre, but with alternate ridges and gutters converging towards the centre, as if some contracting process had occurred which caused a puckering of the conjunctival covering.

The four sides of the tumor were covered by conjunctiva, which was materially altered in its structure, particularly on the upper surface. The base of the tumor was generally flat, though somewhat uneven from adhesion of some of the connective tissue and muscular structure of the lid. The growth was moderately hard and resisting to pressure, somewhat elastic and yet brittle. Unfortunately the specimen was placed in Müller's fluid before the iodine test for amyloid degeneration had been applied. But after it had lain for an hour, it was taken out and carefully washed, and then a watery solution of iodine was applied to a freshly cut surface. It gave the usual reaction in part, that is the reddish-brown color appeared in spots almost immediately, but the blue or violet reaction was only faintly apparent after some minutes, and was not especially intensified by the addition of sulphuric acid. It was then hardened at first in Müller's fluid, and afterwards in a solution of two parts of alcohol to one of water, and shrunk somewhat in the process. Sections were made vertically through the tumor, in a direction from before backward. The conjunctiva was found to extend over the entire surface and sides. The conjunctival surface was everywhere covered by its epithelium, which however varied very much in thickness in different parts. On the anterior and posterior margins of the growth, where the surface passed over into the sides, the epithelium was flattened out to a very thin covering, which in places consisted of only a single layer of cells, which were placed almost end to end, and looked like fibres with nuclei. In the hollows or gutters of the upper surface

the epithelium was very much thickened, there being in some places as many as eight or nine layers of cells. The deeper cells were club-shaped, cylindroid, or almost spherical, while the upper cells were flattened out like tesselated epithelium, and the intermediate layers were modifications of the deep cells, which gradually approached the superficial cells in form.

Immediately beneath the epithelium, especially in the hollows of the upper surface, the tissue was the true small, round-cell sarcoma, unpigmented, with some fusiform cells distributed very unequally, and a few small blood-vessels.

A little deeper the true sarcoma tissue cells were arranged in ovoid groups, without there being any distinct alveolar arrangement, and here were met a few large, round and ovoid cells, with one or more nuclei. There was in this region some little development of connective tissue, and the vascular development was more marked, but there was but very little amyloid infiltration.

Still deeper there appeared an extensive infiltration of waxy-looking, colorless, irregular masses, varying very much in size, entirely structureless, sometimes isolated, and then generally small, while in other places there were large, irregular masses, as if from the coalescence of numerous smaller ones. These nodules of infiltration were not colored by either carmine or hæmatoxylin. The nearer the centre of the growth was approached, the larger became the masses of amyloid infiltration, until all sarcoma cells, both round and fusiform, and all connective tissue structure disappeared, and there was nothing to be seen but amyloid matter entirely colorless.

Still deeper towards the base of the tumor, the small, round, nucleated cells of the sarcoma tissue began to reappear, with more or less connective tissue formation, until the muscular and subcutaneous structure of the lid was reached. In the region of pure amyloid infiltration, the vessels were more numerous, running sometimes between the amyloid deposits, and thus forming irregular alveoli for the infiltration. The walls of the smaller arteries and capillaries in places showed well-marked signs of infiltration, which gave

a glassy appearance to the vessels. The internal coat seemed to be the one first affected, but the muscular coat also had the homogeneous, glistening, indistinct appearance, and in some cases the external coat or adventitia was infiltrated. In the centre of the growth the connective tissue immediately surrounding the vessels was also involved, and here it could be seen that the infiltration pressed from within outwards. The walls of the vessels being thus involved, their calibre was diminished, which would tend to produce anæmia of the part; and the effect was, no doubt, increased by the pressure of the infiltration outside of the vessels in the surrounding tissue.

ON THE USE OF DUBOISINE.

By Dr. W. W. SEELY, of Cincinnati, O.

THE experiments of M. Hubler have quite settled that *Duboisine*, the active principle of the plant Duboisia Myoporoides, in its general action, is quite similar to that of atropine, only much more powerful. The experiments of Drs. DeWecker and Galezowaki demonstrated its action upon the pupil and muscle of accommodation. They have stated that it dilates the pupil and paralyses the ciliary muscle more rapidly and powerfully than atropine, and that its effects are less lasting. Dr. Wecker likewise states that its effects upon the conjunctiva, are less irritating than those of atropine.

In view of such claims for the new mydriatic and the room left vacant by atropia, greater or less according to individual experiences, everything in connection with duboisine will certainly be of interest.

I began the use of duboisine in December, 1878, its extremely high price entirely precluding, of course, my putting it into the hands of patients, with a few exceptions. From the beginning I have used a 4 grain solution (gra. iv ad aq ℥ i) instilling several drops at one sitting, now and then using it twice in both eyes within half or three quarters of an hour. Its effects upon the pupil and ciliary muscle would occur in from 4 to 7 minutes, and in quite a large number of patients without producing any unpleasant symptoms. Its action as a mydriatic and paralysant of the ciliary

muscle is marvellously prompt and so far as I have seen no irritation is produced upon the conjunctiva. In four cases of iritis in which atropia was complained of a good deal by the patients, duboisine acted in the kindest manner. I have only had the opportunity of substituting duboisine for atropia in one marked case of idiosyncracy to the latter. Some time ago I published (*Lancet* and *Clinic*) a very severe case of irritation from atropia in a patient I had operated upon for cataract. It caused great irritation of the conjunctiva, a frightfully annoying condition of the throat, nose and mouth. One result of the irritation upon the lids was the formation of acute chalazia. The clearest opening in the capsule was almost covered by the lower margin of the pupil, so mydriasis was necessary for good vision. The strength of the solution was reduced till it was just sufficient to keep the pupil dilated for a day or two at a time, and yet the patient continued to complain a great deal. Not being ready for an after operation, the patient continued the use of the atropia for over a year, till I put in some duboisine. In order to test it thoroughly I used about three drops of a 4 grain solution *in both eyes*. On his return the next day he said he did not think he felt any effect whatever, and I wrote for a one grain solution, which caused no sort of inconvenience.

I may say that so far I have not seen a case where duboisine was anything but soothing to the conjunctiva, nor have I seen any injurious effects in any way upon the eyes, but my experience has not been so pleasant as regards its constitutional effects. Some time ago I published a case of "marked susceptibility to duboisine" (*Lancet* and *Clinic*), the first case I had seen at that time, and as I had only used about 3 drops of a four grain solution, in *one eye*, while I had been in the habit of using 4 or 5 drops in *both eyes* without any inconvenience, and found some of the physiological symptoms of the drug manifest in five minutes, extreme dilatation of the pupil, ciliary paralysis, *great vertigo*, and "drowsiness," and as the same patient had used several months previously a 4 grain solution of atropia three times a day for two or three weeks, I felt justified in regarding the

above title applicable. I found afterwards that even a one grain solution in this patient caused a slight "queer feeling in the head" as the patient expressed it. Since publishing this case I have had one other very marked case of vertigo, and at least two or three less marked.

What I have written applies only to a 4 grain solution rather freely instilled, occasionally twice at the same consultation in both eyes. I have had one case that required at least two instillations at separate consultations to bring out the entire refraction.

All of my cases thus far have shown a state of mydriasis of much shorter duration and of less annoyance than is caused by atropia.

As to the action of duboisine upon the fundus, in regard to which I have made quite a number of ophthalmoscopic examinations, before and after its use, I can report nothing definite. My experience as far as it goes shows that a four grain solution of duboisine may be used with impunity, at least when there is no diseased state of the fundus, and that vertigo is quite liable to occur even when great care is taken to prevent escape into the tear sac. I fancy a three grain solution will answer any purpose, and a two for all ordinary purposes, and that by the latter the liability to constitutional symptoms will be very much lessened if not done away with.

Of course weaker solutions will destroy some of the brilliancy of its action, which certainly is something wonderful when the drug is used as largely as I have done.

I think the promptitude of action of duboisine, and its apparent freedom from irritation upon the conjunctiva demand for it a thorough test.

THREE CASES OF SUCCESSFUL REMOVAL OF SARCOMA OF THE IRIS.

By H. KNAPP

TO the limited number of cases of malignant tumors of the iris which are on record, I can now add three new cases, which are of importance not only on account of the nature of the growths, but also on account of their successful removal with preservation of the eyes.

In the *first* case—that of an Irishman, æt. 36, on whom I operated four years ago—the tumor occupied fully one third of the iris. It was white, slightly vascular, and obstructed the whole pupil when not artificially dilated. There were no symptoms of irritation. The background was normal, and $S=\frac{20}{70}$, when the pupil was dilated. The removal of the growth by a peripheral section in the sclero-corneal junction was thorough, but followed by a moderately intense iridocyclitis, with a slightly receding scar. In about six weeks after the operation the eye was free from any irritation, and remained so as long as the patient was under my observation, which was about a year. His sight was $\frac{20}{200}$. The tumor consisted of white spindle cells, closely packed together. It originated in the stroma of the iris, without a definite line of demarcation.

The *second* case was that of a French woman of 35 years of age, who had a blackish tumor, the apparent size of a small pea, in the lower part of her brown iris. It had been there for many years, its growth being very slow. For three or four years there had been repeated attacks of iritis

in that eye, leaving posterior synechiæ. The last attacks, which I witnessed, were accompanied by turbidity of the ocular media and increase of tension. They, however, yielded to antiphlogistic treatment, leaving the vision and eye in its previous condition. $S=\frac{20}{100}$; *F* complete. There were in that eye several circumscribed opacities of the lens, evidently congenital.

I removed the tumor with the corresponding piece of iris. The wound healed kindly; the sight was as good as before the operation. During the three years that have elapsed since the operation, only two very mild attacks of iritis have taken place, which disappeared in a few days.

The structure of the growth was a melanotic spindle cell sarcoma, with a considerable admixture of fibrous intercellular substance. It grew from the stroma of the iris without a definite boundary line, but also without infiltration of the neighboring tissue.

The *third* case came under my observation lately, and since it is a typical specimen of the disease under consideration, I may be pardoned for reporting it in detail:

William Taylor, æt. 22, of New York, presented himself to me at the N. Y. Ophthalmic and Aural Institute, in November, 1878, stating that ten years ago a small speck was noticed in his left eye. He felt no inconvenience in his eye until four years ago, when from time to time there appeared a veil-like obscuration of the visual field, lasting the whole day, but disappearing when he could sleep during the day. Last winter the obscurations were more frequent, and accompanied by ocular and circumorbital pain, injection and lachrymation. During the last month he had four or five attacks, accompanied by halos. They rendered him almost blind at the time, and caused violent pain and lachrymation.

Stat. pr. Exterior, *T*, *S* and *F* normal. On the outer and lower portion of the iris, just below the horizontal meridian, a tumor, arose from the outer two-thirds of the iris, projected into the anterior chamber and encroached upon the pupillary area as far as its centre. It was yellowish-red in color, and studded with a number of red dots and small lines. It was not quite so large as a small wild strawberry, was oval in shape, the apparent longest diameter being about 3‴, the transverse diameter 2‴. The pupil

was clear and reacted freely; media clear; low degree of retinal congestion; retina adjacent to disc slightly veiled.

Although urged to allow the operation to be done immediately, he waited until December 11th, 1878. A week previously he had a severe glaucomatous attack. The tumor appeared larger, and projected somewhat farther into the pupillary space than at the first examination. Slight conjunctival injection.

Removal of tumor.—A narrow-bladed cataract knife was thrust into the outer sclero-corneal margin, near the horizontal meridian, passed between tumor and cornea without wounding the tumor, and pushed out near the lower margin of the cornea.

ANATOMICAL EXAMINATION OF THE TUMOR.

The specimen was examined after hardening in Müller's fluid for six weeks, and in absolute alcohol for a few days. It then measured 3.5 *mm.* in length, 2.5 *mm.* in breadth and 2 *mm.* in thickness. Sections through the embedded specimen showed that the growth originated with a broad basis in the tissue of the outer and middle portions of the iris, without, however, reaching the outer, and still less the inner periphery of the iris. Over its basis it broadened, so as to lie as an egg-shaped tumor upon the iris. Its structure was uniform throughout, appearing, when moderately magnified, regularly granular, white, with some pigment dots, and many blood vessels. Its basis reached to the uveal pigment, the other layers of the iris being in that part completely replaced by the tumor, the limits of which towards the neighboring tissue were tolerably well defined. Examined under high powers, the tumor was composed of a well marked, finely granular and delicately striated basis substance, which was crowded with round and slightly oval cells, the large nuclei of which were surrounded by thin layers of protoplasm, not always nicely distinguished from the basis substance. The nuclei were granulated, the nucleoli only moderately conspicuous. The size of the round cells exceeded that of white blood corpuscles, having from two to three times the diameter of the latter. The appearance of the cells was remarkably uniform throughout the whole tumor, only in several places the cells were spindle-shaped, without change in the size and character of the

nuclei. Some of the blood vessels were distinguished by the thickness of their walls, especially the muscular coat, which in the cross section showed several layers of smooth muscular fibres. This struck me as being unusual, since the sarcomata of the ciliary body and choroid have vessels with very thin walls. The tumor of the iris was in this respect distinguished by the same peculiarity as the normal iris, unusual thickness of the muscular coat of its blood vessels. The surface of the tumor showed no difference from its interior.

The parts of the iris adjacent to the tumor were, to a certain extent, infiltrated with nuclei, like those in the tumor, so that the growth had neither a capsule nor a well defined boundary line.

From this examination it is evident that the growth was an unmistakable specimen of sarcoma of the iris. Its structure, and the infiltration of the adjacent parts, prove that it was a malignant tumor. Its slow increase during the first years, its rapid increase and the repeated glaucomatous attacks during the last six months, are in conformity with the usual course of intraocular sarcoma. Without removal of the pseudoplasm, the destruction of the eye would have been certain, and the infection of the organism, with ultimate fatal termination, only a question of time.

The whole history of this case is, in every feature, similar to the case successfully operated on by Dr. C. J. Kipp, and described in these ARCHIVES.* As to the literature on sarcoma of the iris, I may refer to Dr. Kipp's paper, adding only that there are three more cases on record than mentioned by Kipp. These cases are, one by Lebrun,† of very rapid growth. The tumor was removed by iridectomy, after which the pre-existing inflammation increased in severity, and a new growth protruded through the wound, necessitating the removal of the globe. The sarcomatous nature of the tumor is, perhaps, somewhat doubtful, since a syphilitic process could not fully be excluded. Another case is described by Dr. D. B. St. John Roosa, of New York,‡ and

* Vol. V., p. 34, etc. † Annal. d'Ocul., Vol. LX., p. 209, 1869.
‡ Trans. Am. Ophthalm. Soc., 1869.

illustrated by a beautiful colored drawing. Though the patient left this city without being operated on, I do not doubt the diagnosis of the tumor, having personally examined the patient when Dr. Roosa presented her to the N. Y. Ophthalmological Society. A third case is that of R. B. Carter, of London.*

There were sarcomata in both irides. In removing the tumor, Carter devised an original procedure. He thrust a broad needle with parallel blades on one side of the tumor into the anterior chamber, left the needle in, in order to keep aqueous back and steady the globe. Then he made with a lance-shaped knife, held with his other hand, an incision into the cornea on the other side of the tumor. After withdrawing both instruments, he divided the bridge between the two openings with a canaliculus knife, and extracted the tumor by means of a (Tyrell's) blunt hook. The operation was unsuccessful, for C. adds: "In this particular case the character of the growth rendered the operation of no avail, but the method pursued is one which may be useful in more favorable cases."

It seems to me that a lance-shaped knife in cases of small tumors of the iris, or a narrow-bladed extraction knife in cases of larger tumors, is the most suitable instrument. The incision should be large enough to make the tumor and the adjacent part of the iris prolapse on pressure upon the scleral lip of the section. Moreover, the incision should be curved, as in the flap extraction of cataract, and not linear, since the linear wound has too little tendency to gape, and consequently offers a greater obstacle to the easy protrusion and complete removal of the tumor than the curved section, and on the other hand is more apt to retain the edges of the iris between its lips, leading to imprisonment of the iris and its consequences. Care should be taken during the passage of the knife through the anterior chamber not to wound the tumor, lest bleeding into the anterior chamber render the further steps of the operation uncertain. In every case, but especially when hemorrhage occurs, it seems advisable to try

* Trans. Lond. Clin. Soc., Vol. VII., and Carter's text book, Am. Edit., 1876, p. 273.

to make the tumor protrude by pressure on the posterior lip of the wound, and then to grasp the whole base of the protruded part between the blades of a strong pair of forceps, the branches of which are toothed not only at their ends, but to the extent of 4 or 5 *mm.* Since these tumors seem to originate in the outer and middle thirds of the iris it is, of course, of importance to exsect the iris as peripherically as possible. Yet the section should not be situate too near the equatorial border of the sclero-corneal junction, in order to prevent prolapse of vitreous and hyalitis or cyclitis. In the first case, reported above, the section was very peripheric, and cyclitis was the consequence. The safest locality for the section, it seems to me, is in the sclera-corneal junction, near the transparent margin of the cornea.

MARCH 17th, 1879.

THE NEW CONSTANTS OF THE HELMHOLTZ DIAGRAMMATIC EYE.

By ALBERT G. HEYL, M. D., of Philadelphia.

IN the *Archiv für Ophthalmologie* for 1874, there is an article entitled "Resultate einiger Ophthalmometrischer und Mikrooptometrischer Messungen," written by Dr. Reich, formerly a pupil of Helmholtz. In this article a table is given containing, according to the author, the new constants of a diagrammatic eye as accepted by Helmholtz. Stammehaus, in his "Dioptrik des Menschlichen Auges," in working out the formulæ for some of the constants, seems to arrive at somewhat different results, and Nagel, in a recent review, refers to the discrepancy in the following words: "Man enfährt jedoch nicht warum der Verfasser in einigen zahlen von den Helmholtzschen, wie sie authentisch von Reich publicint sind, abweicht." Having had occasion during the past year to use the diagrammatic eye, I found it necessary to examine into the correctness of the table given by Reich. I propose in this paper simply to give the result of the examination.

The constants of a diagrammatic eye may be divided into two classes:

1. Those ascertained by direct measurement or inspection. Such are, for example, the radius of corneal curvature, the indices of refraction of the media, etc. These, as given in Reich's table, differ in some respects from those formerly accepted by Helmholtz. I shall assume for the present that they are correctly given.

2. In this class belong the constants which are ascertained by calculation; as, for example, the foci of the cornea, lens, entire eye, etc. It is among the members of this class that the discrepancies already alluded to occur. I propose to take the members of this class one by one, and to examine into the correctness of

the calculation. If the latter be found correct, it will be simply so stated without going into details; if, on the other hand, suspicion exist as to any constant, the calculation will be carried out in full, and the correct result be given. We commence, then,

1. With the foci of the cornea. According to Reich, the focus anterior is 23.266 *mm.* from the vertex of the cornea, and focus posterior 31.095 *mm.* These numbers are correct, and agree with those given by Stammehaus. Let us then, without delay, pass on to consider

2. The focal distances of the crystalline lens, the anterior of course being equal to the posterior. Reich gives for these 50.671 *mm.* instead of 43.707 *mm.* as formerly accepted by Helmholtz; Stammehaus gives 50.6171 *mm.* There is a discrepancy here of 0.0539 *mm.* Let us examine into this in detail. The formula as given by Helmholtz[3] reads:

$$F_1 = F = \frac{n_1 n_2 r_1 r_2}{(n_2 - n_1)[n_2 (r_2 - r_1) + (n_2 - n_1) d]}$$

I shall give the values of the different constituents of this formula, and then proceed to work out the calculation.

These values have been taken from Reich's table, and belonging to the first class of constants, as already stated, are assumed to be correct; *d*, strictly speaking, ought to be placed in the second class, but no doubt can be entertained of its value.

$n_1 =$	1.3365	Log. 1.3365 =	.1259690
$n_2 =$	1.4371	" 1.4371 =	.1574870
$n_2 - n_1 =$	.1006	" .1006 =	$\bar{1}$.0025980
$r_1 =$	10.	" 10 =	1.
$r_2 =$	−6	" 6 =	.7781513
$d =$	3.6	" 3.6 =	.5563025

Substituting, we have:

$$F_1 = F_2 = \frac{13365.\quad 14371.\quad 10.\quad -6}{1006\ [14371. - 16 + 1006.\quad 3.6]} \text{ or}$$

$$F_1 = F_2 = \frac{13365.\quad 14371.\quad -60}{.1006. - 22.63144}$$

Substituting the logarithms:

.1259690	1.3547121 =	Log. of 22.63144
.1574870	$\bar{1}$.0025980 =	" .1006
1.7781513	.3573101	
2.0616073 =	Log of numerator.	
0.3573101 =	" denominator.	
1.7042972 =	" 50.6171.	

F_1 and F_2, therefore, are equal to 50.6171 *mm.*, the same result as given by Stammehaus.

3. We come next to the principal points of the lens. Reich gives 2.12 *mm.* as the distance of the first principal point from the anterior surface of the lens, and Stammehaus gives 2.126 *mm.* for the same. As the difference between the two consists mainly in the calculation being carried out to the third decimal place, we pass it without further remark. The distance of the posterior principal point from the posterior surface of the lens, as given by Reich, is 1.274, *mm.* and by Stammehaus as 1.2756 *mm.*; here a discrepancy exists of 0.0016 *mm.* Let us briefly examine into this. The formula[3] reads:

$$h_2 = - \frac{n_1 \, dr_2}{n_2 (r_2 - r_1) + (n_2 - n_1) \, d}$$

Substituting as before, we have:

$$- \frac{1{,}3365 . \; 3{,}6 . \quad -6}{1{,}4371 . - 16 + {,}1006 . \; 3{,}6}$$

Substituting the logarithms, we have:

.1259690
.5563025
.7781513
1.4604228= Log. of numerator.
1.3547121= " denominator (*vide* preceding paragraph).
.1057107= " $h_2 . h_2 = -1.27558$

This is practically the same result as obtained by Stammehaus.

4. Let us now turn our attention to the complete eye.

For the anterior focal distance we have 15.5025 *mm.* as given by Reich, 15.4983 *mm.* as given by Stammehaus. The formula[4] is:

$$F_1 = \frac{\varphi_1 f_1}{\varphi_1 + f_2 - d}$$

f_1 and f_2 represent the focal distances of the cornea. φ_1 represents the anterior focal distance of the lens. d represents the distance of the first principal point of the lens to the second of the cornea; it is therefore equal to 2.126 *mm.* +3.6 = 5.726 *mm.* Substituting, we have:

$$F_1 = \frac{50.6171 . \; 23 \; 266}{50.6171 + 31.095 - 5.726} \quad \text{or} \quad \frac{50.6171 . \; 23.266}{75.9861 .}$$

Introducing the logarithms, we have:

1.7042972= Log. of 50.6171
1.3667212= " 23.266
3.0713184

1.8807341= " 75.9861
1.1902843= " 15.4983,

Let us now turn to the calculation of the posterior focal distance of the complete eye. The formula[5] reads:

$$F_2 = \frac{\varphi_2 f_2}{\varphi_1 + f_2 - d}$$

Substituting, we have:

$$F^2 = \frac{50.6171.\ 31.095}{50.6171 + 31.095 - 5.726}$$

Substituting the logarithms:

1.7042972= Log. of 50.6171
1.4926902= " 31.095
3.1969874= " numerator.
1.8807341= " denominator.
1.3162533= " 20.7135=F_2

These results agree with those given by Stammehaus.

5. We have to consider, in the next place, the principal points of the entire eye. We are to determine, first, the distance h_1, or, in other words, the distance of the first principal point of the combined system, from the vertex of the cornea. Secondly, the distance h_2, or, to put it in other words, the distance of the second principal point of the complete eye, from the second principal point of the lens. The formula[6] for

$$h_1 = \frac{d f_1}{da - \varphi_1 - f_2} = \frac{5.726.\ 23.266}{5.726 - 50.6171 - 31.095}$$

.7578513= Log. of 5.726
1.3667212 " 23.266
2.1245725= " numerator.
1.8807341= " denominator.
0.2438384= " 1.7532 = h_1 = − 1,7532

$$h_2 = \frac{d \varphi_2}{d - \varphi_1 - f_2} = \frac{5,726.\ 50.6171}{5,726 - 50,6071 - 31,095}$$

.7578513= Log. of 5.726
1.7042972= " 50.6171
2.4621485= " numerator.
1.8807341= " denominator.
.5814144= " 3.81429 −3.81429=h_2

6. There remain to be shown a few constants which depend for their accuracy on the preceding calculations. These are the loci of the focal points, principal points and nodal points. As the

calculation of them is a matter of simple subtraction and addition, I shall simply place the results in the following table, which is intended to show, side by side, the numbers given by Reich and those which have been obtained in the preceding work. The first column of figures are those given by Reich, and the second those which I have obtained. When no numbers are given in the second column, it will be understood that my results have been the same as those in the first column :

	Reich.	Heyl.
1st class of constants.		
Index of aqueous and vitreous humors and corneal moisture.	1.3365	
Index of refraction of lens (total)	1.4371	
Radius of corneal curvature	7.829	
Radius of ant. surface of lens	10.	
" post. " "	6.	
Locus of ant. surface of lens	3.6	
" post. " "	7.2	
2nd class of constants.		
Anterior focal distance of cornea	23.266	
Posterior " "	31.095	
Focal distance of crystalline lens	50.671	50.6171
Distance of anter. princip. point of lens from ant. surface	2.12	
Distance of post. princip. point of lens from post. surface	1.274	1.2756
Distance of principal points from each other	.206	2.04
Anterior focal distance of eye	15.5025	15.4983
Posterior " "	20.719	20.7135
Locus of anterior focus	13.7525	13.7451
" posterior "	22.834	22.8236
" 1st principal point of eye	1.750	1.7532
" 2d " " "	2.115	2.1101
" 1st nodal " "	6.966	6.9684
" 2d " " "	7.331	7.3253

We have thus reviewed in detail this table of constants, and it will be seen that my results on the whole agree with those of Stammehaus. An apology might seem necessary for the detailed manner in which the formulæ have been worked out, but I am persuaded that it will not so appear to any one whose misfortune it has been to toil through pages of calculation only to find his data incorrect and his labor wasted. The most considerable error is in the focal distances of the lens, and the similarity of figures in the different results might well lead to the supposition that a

typographical error exists in Reich's table. The other errors are indeed quite small, but it requires no argument to prove the need of accuracy in every constituent of a table of this kind. Finally, this paper has been based on the assumption of the correctness of the first class of constants as given by Reich; this assumption is probably just; if not, it simply shows another error in the table. It is not for me, however, to substitute the values which I have given in the table of constants, but it may be hoped that in the near future the renowned author of it may give to the public, under his own name, a table bearing the results of his latest investigations.

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ECTROPION TREATED BY TRANSPLANTATION OF FLAPS WITHOUT PEDICLE.

By Dr. JOSEPH AUB, of Cincinnati.

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INJURIES of the eyelids from burns, contused wounds, caries of the orbit, etc., have their greatest danger in the results of the cicatricial contraction following the reparative process. The ectropion of the lids, combined as it frequently is with an inability fully to close the palpebral fissure, and thus give the eye the natural protection which it so much requires, is a deformity at times, so painful and hideous, as to bring the patient to submit to almost any procedure for relief. Various plans have been recommended for this condition including the twisting of flaps, the transplantation of flaps and the sliding and stretching of flaps, but all have the disadvantage, that if union by first intention does not take place, the deformity resulting from the operation is greater than it was before.

Within the past five years I have performed blepharoplasty seven times for this condition, three of these cases being caused by burns from fire, two by burns with chemicals, one by a burn with hot metal in the conjunctival sac and on the face, and one resulting from caries of the orbital margin. The flaps in these cases were always taken from the parts surrounding the eye, either forehead, temple or cheek, and the methods employed included the twisting of the flaps, as well as the sliding of the same as recommended by Knapp. In the different operations the results were never entirely satisfactory to me, for if the flaps healed as

desired, they remained swollen and discolored, and there was an annoying cicatrix in the situation whence the flaps were taken. Or if the flaps did not unite per primam or even if sloughing of part or the whole of the flap occurred, we had the additional mortification of having increased the disfigurement without any compensating benefit. Under such circumstances, the operations made by Wolff and reported in the *London Medical Times and Gazette* of June 3d, 1876, as well as the one reported by O. F. Wadsworth, M.D., in the *Boston Medical and Surgical Journal* of December 28th, 1876, were welcomed as giving us something new which promised to do away with all the defects of the other operations. The placing of small skin-grafts on a suppurating surface after failure of union in a flap, I had resorted to in one case, but to attempt to place *a large flap without any pedicle upon a fresh surface* was an immense stride forwards. In 1877, I had a little patient under observation and occasional treatment, for ulceration of the cornea resulting from exposure of the eye in consequence of ectropion. I proposed to her parents the new plan of treatment, and readily obtained their consent.

The patient, Lilly McCarthy, aged 4 years, was burned severely in the face when nine months old by falling against a hot stove. The cicatrix resulting prevented the lids from being closed, and about three months afterwards the parents consulted me as to what had best be done to protect the eye. From the exposed condition of the parts, the eye was in a continuous state of irritation. The patient was very weak and anæmic, and its life despaired of, so that at the time palliative remedies only were recommended. The eye was covered with a hollow shell made of pasteboard, which warded off the external irritants and shaded the eye from the light. For the corneal trouble existing at the time, sulphate of atropia was used. The ulceration healed, leaving a very small opacity. In the same manner the eye was attacked three different times, each fresh ulceration causing a fresh opacity. The parents, therefore, readily consented to a procedure which promised to relieve the exposed condition of the eye, and that without any increase of the deformity. The health of the patient was excellent; she had developed from a weak, puny infant to a strong, healthy child, whose only complaint was the annoyance of the eye.

The condition of the eyes at the time of the operation was as follows (see Figs. 1 and 2): When the eyes were opened there was a convergence of about 2''' (Fig. 1). The slightest effort upon the part of the patient to look down or close the eye caused a complete and persisting eversion of the upper lid (Fig. 2), exposing the entire tarsal conjunctiva. Whilst playing, the child would run against this conjunctival surface, causing it to bleed freely. The cornea was entirely exposed during sleep, unless by a forcible effort the upper lid was *everted*, and by an upward movement the upper portion of the cornea was brought under the upper lid. The lower lid of the right eye was drawn downwards by cicatrices in the cheek, pulling the lid away from the eyeball, especially downwards and outwards. In this trough the tears were continually collecting, and then flowing over, caused an excoriation of the margin and the outer surface of the lid. The conjunctiva was swollen and injected. The margin of the lid was elongated.

The right side of the forehead was cicatricial from a point about ½'' from the median line to the temporal region. The outer ⅔ of the eyebrow was almost destroyed and drawn upwards and outwards. The skin of the upper eyelid was drawn upwards by this contraction, and shortened but not thickened. From the eyebrow, at the

FIG. 1.

FIG. 2.

CONDITION BEFORE OPERATION.

union of the inner and middle third, a dense cicatrix, binding the skin to the orbit, extended downwards to the lid margin, at a point immediately over the punctum. When the patient looked downwards eversion immediately followed, and the parts returned to their position only after the patient looked upwards.

The patient having been chloroformed, with the assistance of Prof. W. A. Rothacker, I proceeded to operate. The incision was made, commencing at the inner orbital margin, about half way between the margin of the lid and the remaining portion of the eyebrow, and extended in an irregular manner outward through the dense cicatricial band and to the lower border of the burned eyebrow. The thin skin on the outer portion of the eyelid was brought down with the lid margin, and all the parts freely dissected loose until the lid margins could be brought in apposition without any traction. They were united by three strong sutures. The surface thus exposed measured 1⅝″ by ⅞,″ and bled freely only a short time. To cover this defect, it was determined to remove a portion of the skin from the inner surface of the left arm over the biceps. The flap taken thence measured 2⅝″ by 1⅜″, and was carefully prepared by cleansing its inner surface from all adipose and subcutaneous tissue. It was then carefully washed in lukewarm water and placed in the defect. At first it seemed as if the flap were too large, and it was found necessary to gather it together in the centre before the edges could be brought to lie in apposition with those of the defect. After a great deal of careful labor this was accomplished ; one suture was introduced in the uppermost point of the flap to prevent the edge from rolling in on itself, and to avoid the sliding down of the flap in any movement of the parts. Goldbeater's skin was used to cover the entire surface of the flap and lids ; cottonwool was spread over all, and a light compress bandage applied. The wound in the arm was closed by dissecting up the side of the defect and bringing the parts together by seven sutures. Adhesive plaster strips were applied to support the sutures.

The compress bandage was removed every day, but the goldbeater's skin was allowed to remain for four days. Through it the condition of the flap could be observed. It looked healthy, and had become perfectly smooth. There was considerable secretion from the conjunctival sac, which caused some difficulty in renewing the bandage. The parts were always carefully cleaned with carbolized water, and in order to prevent the secretion from causing the bandage to stick too closely, a piece of muslin, spread with simple cerate, was laid over the goldbeater's skin, and the compress bandage applied over all. The eighth day the sutures which held the lid margins together were removed. The simple cerate had caused a softening of the epidermis on the flap, and this was

easily washed off with a camel's hair pencil. At the point where the suture was introduced into the flap, a redness and swelling was now noticed, and the flap seemed slightly elevated. At all other points firm union existed, and the edges of the flap bled readily on being pricked with a pin. The suture was now removed, and pus escaped from under the flap. This was the only point of suppuration. From this day on no compress bandage was applied. The goldbeater's skin was again applied over the upper lid, and the point of suppuration soon showed healthy granulations, which were touched with arg. nitr. They were covered with epidermis on the thirteenth day. On the fifteenth day the patient was discharged from the hospital, and allowed to return to her home.

The wound in the arm did not heal *per primam*; the sutures gave way on the third day, and left a large exposed surface. This was, however, soon covered with healthy granulations, resulting in a thickened linear scar.

The patient was kept under observation until December 12th, when the condition was as follows: The ectropion of the upper lid was entirely gone; the movements of the lid were free. The flap had become so intimately united with the surrounding parts that the line of union could only be recognized after the closest scrutiny. The flap showed one point of thickening about the size of a split pea at the point of suppuration. The size was 1⅛″ by ⅝″. The lower lid was still in the same condition, and the palpebral fissure could not be completely closed on account of the cicatrices in the cheek preventing the close application of the lower lid to the margin of the upper. I desired to remedy this defect in the same manner as in the upper lid, by transplantation of a flap, and to shorten and lift the lid by excising a triangular piece of the lid at the outer angle in combination with tarsorraphy. But the parents would only consent to the latter portion of the operation, preferring to have another operation if the result was not satisfactory.

Assisted by Dr. Rothacker, this was done on the same day. Union was perfect, and, *per primam*, the patient was discharged at the end of the fourth day. The result was as good as could be expected (Fig. 3), although the cicatrix in the cheek still prevents the close application of the lid margins. A small slit about ⅛″ wide, and ¼″ in length, remains in the centre of the palpebral fissure. The eyes remain closed in sleep. Ectropion of both lids is completely relieved, and the cornea is totally covered. There is no epiphora

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and the injection and swelling of the conjunctiva have disappeared. The patient can perfectly close the eye by forced contraction of the orbicularis; eversion of the lid does not take place even if she looks altogether downwards (Fig. 4). She attends school, plays in the open air, and exposes herself in all winds and weather without experiencing the least difficulty.

FIG. 3.

FIG. 4.

CONDITION AFTER RECOVERY.

In this case we observe the perfect union of the flap edges, *per primam*, and the suppuration taking place at a period only when a suture had been introduced. The flap had been carefully cleansed and washed, so that the subcutaneous tissue was certainly as cleanly removed from one point as from another. That suppuration should have occurred at that point, and only there, I therefore judge to have been caused by the irritation produced by the suture, and by allowing it to remain for eight days before removal.

In May, 1878, Dr. John Corson, of Middletown, asked me to see a case with him, in which the deformity produced was even greater than in the case just described. Gratified by the favorable result of the operation in my case, he determined to try the same method. The description of the case, with the manner of operation, is given below in the doctor's own words:

The patient, Mary C., was injured about six years ago, by falling into the fire, burning the upper part of her face severely. In the cicatrization the skin of both upper eyelids were involved, the left more than the right. The appearance, when the patient

presented herself before me for relief, was as follows (see figure 5): The forehead was traversed by numerous cicatrices, binding down the skin to the underlying parts. The left upper eyelid was entirely destroyed, except the entire tarsal margin and the skin for the width of about ⅛″. The margin was firmly united by cicatrices to the superciliary border, causing an eversion of the entire tarsal cartilage and exposure of the retrotarsal fold. The conjunctiva was swollen and injected, especially that of the everted eyelid. The eye was constantly open during sleep, and the cornea was saved from serious trouble, first, by the contraction of the orticularis, causing the retrotarsal fold to cover it partially, and second, by the forcible drawing of the eye upwards. The right upper eyelid was restricted in its free motions only at the inner third, where a cicatricial band running from the inner end of the eyebrows diagonally downwards to the lid margin, prevented the closing of the palpebral fissure. The band of cicatricial tissue was about ⅓″ wide, and the opening left when the lids were about to be closed was one-half inch in length and one-third inch in its greatest height.

FIG. 5.
CONDITION BEFORE OPERATION

It was determined to remedy the hideous deformity by skin grafting, for two reasons, first, the amount of flap necessary to fill in the defect was too great to be taken from the forehead or cheek, and secondly, the cicatrized skin would not be apt to heal well.

On June 10th, 1878, the patient having been anæsthetized, I proceeded, assisted by Dr. Jos. Aub, of Cincinnati, to operate. The cicatrix holding the left upper eyelid in its position, was divided by an incision, parallel to the lid margin and about one line from it, extending from the inner angle of the lids to a little beyond the outer cauthus. All of the adhesions were divided, and the free margin of the lid attached to the lower lid by three sutures. The raw surface left bled but very slightly. It measured 1⅞″ in length and ⅞″ in width in its greatest diameters. The flap to cover in this defect was removed from the inner side of the right arm over the biceps. It measured 2⅞″ in length by 1⅞″ in

width. After removing it from the arm, all adipose and connective tissue was removed, and after carefully cleansing it in warm water, it was placed in position. To hold it in its place and prevent the skin from rolling in on itself, five sutures were introduced superficially. The whole was then covered with goldbeater's skin, and with an ordinary compress bandage.

The right lid was then operated upon. After dividing the cicatricial band and loosening the adhesions, the lid margin was fastened to the lower lid by sutures. The opening left was ⅞″ by ½″. A flap was taken from the arm immediately above the place whence the first flap was taken, and measuring 1⅞″ by 1″. This was placed in the opening, remaining after dividing the adhesions, the edges coapted, and left without any sutures. Goldbeater's skin covered the entire surface, and the compress bandage was then applied over both eyes.

The arm was treated by dissecting the sides of the defects, and bringing the flaps as far forward as possible, and holding them in position by adhesive strips and a roller bandage. The patient was then given one-eighth of a grain of sulphate of morphia. She did well. Two days after the operation, some free purulent discharge from the inner angle of the left eye. The discharge was from the conjunctival sac. After washing this off with carbolized water, the parts were again covered with a common press bandage.

June 15th. No fever. No swelling; grafts looking healthy. Sutures holding flaps removed. Patient was allowed to sit up in bed.

June 16th. Progressing favorably. Both eyes and arm doing well. Patient out of bed for several hours. From this day on the patient progressed favorably, giving only some slight trouble with her arm, until June 22nd, when the stitches which united the lid margins were removed, and the conjunctival surfaces washed with sol. plumb. acet. The parts were cleansed with carbolized water, and fresh goldbeater's skin applied. There appears to be no suppuration at any point; the flaps have healed without any loss of substance. On the surface the epidermis is scaling off, but was not removed for fear of disturbing the flaps.

June 29th. Eyes were uncovered and allowed to remain so. The goldbeater's skin has formed a scab with the epidermic layer of the grafts, which is dark and hard. This is partially detached, but not removed for fear of rupturing adhesions. Under these scabs the grafts are seen looking healthy.

July 6th. The scabs have been removed showing the surfaces beneath to be perfectly united, and only slightly excoriated on the surface, from the long continued presence of the epidermis. The surfaces measure in the left eye, $1\frac{1}{8}''$ in length, by $\frac{9}{16}''$ in breadth; in the right one, $\frac{11}{16}''$ in length, by $\frac{6}{16}''$ in breadth.

FIG. 6.
CONDITION AFTER RECOVERY.

From this day the eyes improved rapidly. The patient was able to open and close the eyes readily. By the last day of July the new grafts were scarcely to be told from the surrounding tissue, and only one small spot on the left eyelid still showed some roughness. The discharge from the conjunctival surface has ceased entirely, and the eyes look clear and bright. (See figure 6.)

In both cases the flaps removed from the arm were taken much larger than the defect to be closed. Profiting by the experience of Wadsworth, it was determined to allow for a shrinkage of 40 per cent. in the flaps, and in this we were not mistaken. The flaps were all thoroughly cleaned of subcutaneous tissue, and I think that to this fact we can attribute the healing without any sloughing of the parts. The results in both cases are so far superior to anything that can be obtained from any of the other methods of blepharoplasty, that it seems to me this method of procedure will eventually supplant the others in all cases where only the skin has become cicatrized, and the tarsal cartilages with the lid margins have remained intact. That we shall still be compelled to resort to sliding flaps in those cases where a portion of one or both lids has been removed on account of neoplasms, is self-evident.

CONTRIBUTIONS TO THE PATHOLOGICAL ANATOMY OF THE HUMAN EYE.

(Continued from Arch. Ophth. and Otol., Vol. VI., p. 366.)

By Dr. ADOLPH ALT, of Toronto.

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No. XIII.

On the anatomical conditions of the ciliary nerves in an eye which suffered from purulent panophthalmitis; enucleated on account of sympathetic iritis. At the same time an answer to Dr. W. Goldzicher, in Budapest.

Dr. W. Goldzieher's paper on the "Pathological Anatomy of the Ciliary Nerves," (*Zehend. Monatsbl.*, December, 1877), describes besides the compression of the ciliary nerves by accumulations of round cells in the suprachoroidal space, also an infiltration of round cells into the sheath of the nerves, and the fibres of the small branches lying in the suprachoroidal tissue. These facts induced me to examine again in the most careful manner, the specimens which had served me when writing my paper on the anatomical causes, etc., of Sympathetic Ophthalmia, (these Archives, Vol. V., page 395, etc.). The result was the same.

Dr. Goldzieher wants to know, whether my investigations concerned only the largest nerve-branches, and states that in these the microscope can detect alterations with difficulty only. My examinations concerned, and their results therefore, hold good for every ciliary nerve which I was able to find in the examined eye-balls, (in sections through ciliary body and sclera, and after isolation of the nerves lying in the suprachoroidal tissue). I was not satisfied to examine the ciliary nerves surrounded with all the elements of the suprachoroidal tissue, in which state they often appeared to

be in a condition like those described by Goldzieher, but I freed the nerves from their surroundings as much as possible, and moreover, compared the specimens thus obtained with specimens taken from perfectly normal eyes. This mode of examination would, I think, surely have enabled me to find real pathological changes in the tissue of the ciliary nerves, if there had been any.

It appears to me after all, as if Goldzieher had given too little weight to the examination of the *largest* nerve-branches, especially as what he found in the *smallest* nerve-branches induced him to support the idea of a neuritis with intervals of healthy tissue, (sprungweise Neuritis). Now, if he found the same conditions also in the *larger intra-ocular and intra-scleral* branches of the ciliary nerves, they would in his case come nearer to a proof of such a mode of mechanical transmission of the inflammatory process through the ciliary nerves. That such a transmission is possible, I have, however, never denied.

In examining the globe which is to be described below I paid again especial attention to the conditions of the ciliary nerves; the results of this examination will be seen hereafter.

The history of the case is as follows: G. D. C., æt. 29, presented himself at my office on the 20th of November, 1877. He stated that four years previously, at sea (while an officer on board an ocean steamer), his left eye was injured by a cinder. This trivial injury was followed, some days later, by a severe inflammation, and a profuse discharge of thin pus from the eyelids. Since he was at the same time suffering from gonorrhœa of the urethra, he thinks an infection from this source might possibly have taken place. The inflammation lasted several weeks. When it was finally cured, the cornea was opaque, and he had only quantitative perception of light in this eye. The right eye had remained perfectly intact. From that time the injured eye was at times the seat of severe pain, and the right eye seemed to become gradually weaker. Two days before he came to see me he had been to the theatre, and felt at once ("in consequence of the bright light") severe pain in the left eye, in which the right (good) eye soon participated. This condition grew rapidly worse.

The following was the *status præsens* when I saw him first on the 20th of November: *L. E.* The lids are very much swollen; ciliæ are slightly glued together by a muco-purulent secretion. The conjunctiva is very chemotic. The whole of the cornea is undergoing purulent destruction, and the iris can consequently not be seen. The eye is very painful, both spontaneously and upon pressure. *F*—1; *S*=0. *R. E.* The ocular conjunctiva is very much injected, and the iris, which does not appear to be swollen, does not react at all. The background is normal.

I advised immediate enucleation of the left eye. He allowed, however, twenty-four hours to elapse before consenting. When I saw him then, sympathetic iritis was manifest. After the enucleation of the left eyeball, which had nothing remarkable, I had a four-grain solution of atropia instilled into the right eye every quarter of an hour, by day and night. When on the second day after the operation the condition of the right eye did not appear to be in the least changed, I had six leeches applied to the temple, and gave a powerful aperient. Finally, on the third day, the synechiæ began to give way, and on the eighth day the pupil was *ad maximum* dilated and perfectly round. Only very small remains of uveal pigment lay upon the anterior capsule. It took, however, several weeks before the eye was free of repeated relapses of injection. The instillations of atropia were continued until the eye was perfectly free from irritation and the visual acuteness was normal.

The wound caused by the operation healed rapidly, and the iritis in the right eye was at no time accompanied by any other sympathetic affection.

The enucleated eye-ball was hardened in Mueller's fluid. The cornea showed old scars, and a recent infiltration with pus cells which involved two-thirds of its thickness. The whole of the anterior chamber was filled with pus. There was no pupil. The tissue of the iris was very much swollen and infiltrated with round cells. In one place I found a tumor of the size of a pin's-head, consisting of pigmented cells only (melanoma). The posterior chamber, Petit's canal and the anterior third of the vitreous body were filled with pus. The lens was cataractous. The ciliary processes and choroid were filled with round cells and their

overfilled blood-vessels showed white blood-corpuscles in all stages of diapedesis.

The retina as well as the excavated papilla appeared thicker, the optic nerve thinner than normal. With the microscope I found in the optic nerve besides a high degree of hypertrophy of the connective-tissue trabeculæ and atrophy of the nervous elements, which conditions were certainly of long-standing, a large number of apparently recently formed and very hyperæmic blood-vessels, surrounded by larger and smaller parenchymatous hemorrhages. Furthermore, optic nerve and retina were filled with round-cells. The retinal blood-vessels were also in a state of hyperæmia and in the outer granular layer I found a number of cavities filled with fibrinous threads, like those usually found in Bright's retinitis.

It was therefore a case of purulent panophthalmitis, which involved also the retina and all that part of the optic nerve removed by the operation.

And what now was the condition of the ciliary nerves? They showed no pathological change whatsoever. Although the suprachoroidal tissue was filled with round cells and free nuclei, the isolated nerves appeared perfectly normal.

REMARKS. The healing of the wound caused by the enucleation was perfectly normal in spite of the purulent panophthalmitis. It is furthermore evident that the patient owes the recovery of his sympathetically affected right eye only to the speedy enucleation of the eye suffering from purulent panophthalmitis and nobody can possibly doubt, it seems, that the latter was the cause of the sympathetic iritis.

The anatomical examination of the eye-ball showed a very active inflammatory process in the optic nerve and retina, while the ciliary nerves appeared perfectly unaltered.

It seems to me therefore, that this case again tends to support the opinion that the inflammation of eye one may in some cases be mechanically transmitted to the fellow-eye by the optic nerve. I do not, however, want to say more, than that this is *one* of the ways in which sympathetic affections may be brought about, *i. e.* by direct transmission of the inflammation.

No. XIV.

On pathological new-formation of the pigment-epithelium.

On the rare occasion of examining an eye-ball suffering from pigmentary retinitis which was diagnosticated during lifetime and which will be described in the next number of this series, I thought it well to study at first the pathological new-formation of the pigment epithelium. In the following, however, I will only speak of those new-formations of the pigment epithelium, from which the pigmentation of cyclitic membranes or the retina are apt to result.

In case No. 9 of this series I had described a specimen in which, besides other pathological conditions, there was pigmentation of the retina. The pigment lay in that case almost entirely in the outer layers of the retina and its origin could be easily traced to the pigment epithelium. It was, however, impossible to determine exactly in which way the process had gone on. Only a very small number of retinal blood vessels were, as if accidentally, surrounded by pigment. The pigmentation of the retina was the result of chorio-retinitis.

The eye was staphylomatous in a high degree and, as is well known and has already been described by Schiess-Gemuseus, we find frequently in such eyes a new-formation of pigment, starting from the uvea (principally of the pars non-plicata) of the ciliary body and always showing the same typical form, namely, cylindrical off-shoots of the uveal layer, which terminate with a roundish end in the cyclitic membrane or the periphery of the retina, frequently after having given off several branches of the same shape.

The way in which these new-formations are connected with the ciliary body and in which they grow into the tissue lying inwards from the ciliary body, induced not only Schiess-Gemuseus, but also later investigators, to declare them to be blood-vessels with a pigment-covering. The large number of specimens, showing the growth of new-formations, which I have examined, force me to contradict this statement.

In a great many eyes which suffer from some disease of

the uveal tract (especially from cyclitis and staphyloma) the pigment-epithelium cells are partially destroyed—by which means the pigment molecules are freed and may be taken up by the surrounding tissues and blood-vessels—where this is not the case, they often show active new-formations. We then find a hyperplasia of the pigment-epithelium cells, which often forms these tubular offsets, and sometimes we find membrane-like new-formations, and in some cases both modes of hyperplasia are seen. The new-formed cells vary greatly as to their pigmentation and are often free from pigment.

I had formerly often found cell-cylinders in cyclitic membranes, consisting of unpigmented epithelium-like cells, which lay in the cyclitic membrane like the cylinders in an epithelioma, but I never could determine their origin. Lately I have been able to trace them back to the uveal layer of the ciliary body in quite a number of cases, and I have had also occasion to see such cell-cylinders which were partly pigmented and only partly free from pigment. Whilst it is sometimes very difficult to determine that the pigmented cylinders really consist of cells, these can be easily recognized in those cylinders which are only partly pigmented or entirely free from pigment. The cells are more or less conical or cylindrical and lie mostly like radii around a centre, which is, however, not formed by a lumen. The structure of the cylinders is therefore very much like that of the tubular glands or the epithelial cylinders in an epithelioma, and shows equally well in transverse and longitudinal sections,

Whilst in most cases this new formation of tubular excrescences concerns only a small part of the tissue, it seems possible that it leads sometimes to the formation of a tumor consisting of the same element. I have a specimen kindly given to me by my late friend, Dr. F. A. Munson of Albany, N. Y., where numerous tubular excrescences spring from the ciliary body and adjoining part of the choroid, and form such a tumor. The tumor consists of connective tissue which is very rich in cells, and in which pigmented and unpigmented cell-cylinders are embedded. It is to be regretted that by the unexpected death of Dr. Munson, we will probably never hear more of this interesting eyeball.

I have found the membrane-like hyperplasia of the pigment-epithelium layer only in eyes affected with chronic chorioiditis, and only in equatorial and posterior parts of the eye-ball. We find in these cases added to the original pigment-epithelium layer, a number of new layers consisting of cells which vary very much in pigmentation, or are totally free from pigment. In spite of their varying shape, it is evident that these cells are directly produced from the pigment-epithelium. Wherever we find this form of hyperplasia the retina is adherent to the choroid. Rods and cones are mostly destroyed, and the new-formed cells grow into the retinal tissue. Thus the retina is found pigmented in a way which is generally considered typical for genuine pigmentary retinitis, and very similar to the above-named tubular excrescences. Later on, the pigment-cells which have invaded the retina may become isolated from their mother-tissue, and it is then impossible to trace their origin. (Similar conditions are often seen in funnel-shaped detachments of the retina.) In a similar way the retina is pigmented after wounds of this membrane and probably also after isolated ruptures of the choroid, with which, I do not doubt, a rupture of the outer retinal layers is usually combined.

No. XV.

A case of retinitis pigmentosa, diagnosticated before the enucleation.

The eye which is now to be described was removed by Dr. E. Williams of Cincinnati, on the 20th of October, 1876, and kindly given to me for examination by Dr. H. Knapp. Since only a small number of cases of pigmentary retinitis, diagnosed during lifetime, have been thus far examined and described, in which there did not at the same time exist old and important pathological changes in the choroid, I thought it well to report this case.

The few lines by which the eyeball was accompanied, contained the information that the patient, P. N., æt. 64 years, had received an injury of the right eye fifteen years ago. After this the eye had been painful for a long time. When first examined by Dr. Williams, the cataractous lens

was found dislocated into the anterior chamber, and it was thought that this dislocation must have occurred a long time previously to this examination, as the eye had been totally blind for twelve years. Although there was at the time a cataract forming also in the left eye, it was very easy to make the diagnosis of pigmentary retinitis in both eyes. The patient had never suffered from night blindness. He was accompanied by his brother, who also showed pigmentary retinitis in an advanced state. His visual field was very much contracted, and the optic papillæ were very atrophic.

When I received the eyeball it was divided in the horizontal meridian. The longitudinal diameter was abnormally long; the shrivelled cataractous lens lay at the bottom of the anterior chamber. The optic nerve entrance was excavated. The pigmentation of the retina which showed the microscopical appearance of pigmentary retinitis, concerns only a zone of this membrane, about ten lines in breadth and lying equally distant from the ora serrata and the optic papilla. The retina is easily removed from the choroid, there being no pathological adhesions. The pigment-epithelium which adheres to the choroid appears very irregularly distributed. It cannot be discerned whether parts of it are adherent to the retina or not.

Microscopic examination—(*a*). *Surface views of the retina.* Examining the outer surface of the retina I find that in the most peripheral parts of this membrane and where the pigmentation has taken place, the rods and cones are totally destroyed. Near the optic disc they are partially preserved and partially changed into club-shaped bodies. Numerous pigment molecules adhere to this outer surface of the retina. This pigment is granular, not very dark and the molecules form clusters of varying size and shape. There are furthermore a number of pigment-epithelium cells adherent to the retina which have lost their normal shape and are roundish or oval. Besides these pigmented cells there is a greater number of unpigmented ones with a large oval nucleus, easily distinguishable from the underlying cells of the outer granular layer and identical with the unpigmented products of the pigment-epithelium, described in No. 14.

In viewing the inner surface of the retina I find a tough fibrillar tissue, the elements of which are irregularly arranged. The course of these fibres varies greatly in the parts of the retina nearest to the ora serrata and in those affected with the pigmentation. They are more regularly arranged nearer the optic disc. In this part of the retina the nervous fibre layer has nearly its normal thickness, whilst nearer to the ora serrata it is more and more replaced by the hypertrophied connective tissue. Some of the fibres which may undoubtedly be recognized as nerve fibres, are sclerosed, others are in a state of fatty degeneration. There are a number of cavities lying in the tough fibrous tissue at the inner surface of the retina (cysts?), which vary in size and shape. Furthermore I find between these fibres free pigment molecules, and quite a number of round-cells and "seal-ring-cells," such as are frequently seen in the vitreous body. Some of these cells have several nuclei.

The blood-vessels of the retina are represented by tough bands of connective tissue, more deeply stained by carmine than their surroundings and mostly lacking both a lymphatic sheath and a lumen. Some having still a small lumen, filled with blood-corpuscles, show walls transformed into tough connective tissue and thickened (Perivasculitis). Only a very small number of these altered blood-vessels, are here and there surrounded by pigment and pigmentation is evidently not confined to the blood-vessels, as some authors will have it. The number of the pigment cylinders is much larger than that of the vessels of the retina, and under the microscope their arrangement is in no way like that of the latter, which is evident by comparing specimens of this retina with those of an injected normal one.

The pigmentation affects mainly the outer and inner layers of the retina and very seldom reaches as far as the tissue replacing the nerve fibre layer. The pigment is mostly so dark that, like the uveal pigment of the iris, it is often impossible to discern whether it is free or enclosed in cells. In some parts, however, the cells are unmistakable; they have a light round or oval nucleus, which is plainly visible through the pigment. If I follow up such

pigment cylinders under the microscope, I frequently find less pigmented or even unpigmented parts, where these cells are easily recognized. A small number of the pigment cylinders end with a perfectly unpigmented part, composed of cells varying in size and shape, with a roundish or oval nucleus.

(*b*). *Transverse sections of the retina.* Transverse sections exhibit very plainly the reduced thickness of the retina, which in many places is not half that of the normal condition. This is mainly caused by the fact, that all the layers, except the nerve fibre and ganglionic layer, appear to be united into one, consisting of round-cells, similar to the granules and of tough connective tissue. This condition is apparent all over the area of the retina, even near the optic nerve entrance. The two innermost layers of the retina are transformed into a dense connective tissue, in which are embedded the cavities and cells mentioned above, and some ganglion cells in a state of fatty or colloid metamorphosis.

The pigment cylinders, as was seen in the surface views, lie chiefly in the outer layer and but seldom reach the limitans interna. Only very few of the transverse sections show blood-vessels surrounded by a pigment-covering. The latter lies evidently in the lymphatic space surrounding the vessel. That part of the pigment epithelium adherent to the choroid, consists of round and polygonal cells of varying shape. They appear irregularly scattered upon the lamina vitrea. Their pigmentation varies in intensity and there is a great number of cells without any pigment at all.

The other alterations of the eyeball are without interest. Two very small atrophic spots in the periphery of the choroid are of no importance with regard to the disease of the retina.

If I now compare the conditions found in this eye with those described in No. 14, there is no great difficulty in considering the typical pigmentary retinitis as a secondary affection of the retina caused by pathological new-formation (hyperplasia) of the pigment epithelium. If such a hyperplasia of the pigment epithelium is once started, the changes in the retina following it are easily explained. At

first the rods and cones are destroyed, then the new formed cells, partly pigmented, partly unpigmented, grow into the outer retinal layers, where, acting as foreign bodies, they occasion hypertrophy of the connective tissue of the retina and around the blood-vessels (perivasculitis). The same conditions may also be found in chorio-retinitis. In the typical disease, however, which has clinically been styled as pigmentary retinitis, we have a pathological process in the retina caused solely by the hyperplasia of the pigment epithelium, without being combined with or in its turn caused by an affection of the choroid. This is its characteristic feature.

Whilst it was formerly considered that the pigment epithelium was a part of the choroid, it is now generally regarded as a part of the retina. The recent discoveries concerning the purple of the retina seem to prove, however, that the pigment epithelium forms a separate organ, a sort of gland, as was pointed out by Kuehne. We cannot doubt that this gland will become subject to pathological processes peculiar to itself, as other glands are; and the conditions in the eye under consideration, and what has been described in No. 14, seem to prove this statement. It is apparent that the pigment epithelium when in a state of inflammation, has a predilection for the production of tubular effects, although a membrane-like hyperplasia with simple thickening is also observed.

Whether this hypothesis prove correct or not, it is evident from the foregoing description that pigmentary retinitis is not a primary disease of the retina, in which opinion also Leber concurs. (See Graefe and Saemisch, Vol. V, 2, pag. 623, etc.)

We must therefore distinguish between a retinal pigmentation caused by chorioiditis and one caused by hyperplasia of the pigment epithelium without an affection of the choroid.

No. XVI.

On Xanthelasma tuberosum palpebræ.

The following are the results of the examination of five tumors which had been diagnosticated as xanthelasma and

removed by operation. All of the five individuals were females. Three of the cases I examined while in New York, and they were kindly given to me by Drs. H. Knapp and E. Grüning. Two of the cases occurred in my own practice.

The epithelial layer covering the tumors is in all cases thicker than normal. In one case it is perfectly transformed in horny scales. The subcutaneous connective tissue, which is also considerably thicker than normal, consists of very coarse fibres, and has very few blood-vessels. Between the fibres there is a considerable number of cells, filled with fine molecules of a yellowish-brown pigment, and mostly having several offsets. The orifices of the sebaceous glands seem to be all obliterated. The main part of four of the tumors, is composed of large, vesicle-like, round and oval cells with an oval nucleus and several nucleoli. The cell-body can not be tinted by any of the common staining materials, the nucleus assumes only a very light, the nucleolus a darker color. The cells, therefore, appear akin to fat-cells and colloid-bodies. I consider them to be essentially the same and am inclined to believe that they are the metamorphosed epithelial cells of the sebaceous glands. The cells are divided into clusters of varying size and mostly round shape, by bands of tough connective tissue, which show no fatty degeneration, as has been described by some authors.

One of the five cases showed the same pigmented cells in the subcutaneous tissue, but the bulk of the tumor consisted only of dense connective tissue, in which no trace of the glands could be found.

It appears from this that there are two kinds of tumors producing the same clinical picture. One of them seems to take its origin from a hyperplasia of the epithelium of the sebaceous glands. The ensuing irritation and inflammation of the tissue surrounding these glands leads to its hypertrophy and this in its turn to obliteration of the orifices of the glands, which finally causes the retrogressive metamorphosis in the cells of the glands. In the second case we have to deal with a fibrous tumor which while growing destroys the glands entirely.

These two kinds of tumor, so different in their structure, have, however, one thing in common, viz., the pigmented cells which lie in the subcutaneous tissue. I am not prepared to explain their origin; they, however, I do not doubt are the cause of the clinical similarity of the tumors.

According to the literature on this subject, as far as it is at my disposal, there is yet a third kind of xanthelasma, which is characterized by hypertrophy and fatty infiltration of the connective tissue. Waldeyer and others have described this kind in detail. Geber and Simon have mentioned also the tumors which originate in a hyperplasia of the epithelium of the sebaceous glands. The pigmented cells in the subcutaneous tissue do not seem to have been mentioned by these authors.*

More with regard to the literature on this subject may be found in Michel's writings, (Graefe and Saemisch, Vol. IV., 1, § 75,etc.).

No. XVII.

Another case of sarcoma of the choroid with the formation of cartilage.

The eye which is now to be described was enucleated by Dr. Roseburgh of this city, who kindly gave me one half of it for examination. It is greatly to be regretted that no history of the case has been kept.

The part of the globe I had for examination did not include the optic nerve. The posterior part of the vitreous chamber which is bound anteriorly by the detached retina, is filled with a serous exudation. The rest of the globe is occupied by the tumor in such a way, that the latter after having filled the anterior chamber, has pressed the cornea which covers it continuously, very much forward. It thus happened that the part of the eye which lies between cornea anteriorly and ciliary body posteriorly became nearly as large as the remainder of the globe. During the growth of

* Since this paper has been published in the German edition of these ARCHIVES, I received a letter from Professor Waldeyer in which he kindly informs me that he has known and described the pigmented cells "of the subcutaneous tissue" before me, and that he mentioned them especially with regard to the origin of melanotic tumors of the skin. In thus publicly acknowledging his claim to priority on this subject, I beg to thank him for the information.

the tumor the cornea was perforated at its margin, and here the tumor has grown somewhat under the conjunctiva. Iris and lens are wanting.

Microscopically a large number of islets of characteristic cartilage tissue may be recognized. They are in this case all intraocular, are found in all parts of the tumor and with every section new ones are laid bare.

The microscopic examination reveals the fact that the bulk of the tumor is an entirely unpigmented sarcoma. It consists of round-cells and spindle-shaped cells and is traversed by a great many bands of connective tissue. The detached and atrophic retina adheres to the posterior surface of the tumor. The posterior third of the choroid is atrophic; the pigment epithelium here is proliferating, and its cells extend into the serous exudation above mentioned. The anterior parts of the choroid as well as the ciliary body are filled with the elements of the tumor.

The interesting feature in this case are the numerous islets of cartilage tissue. The cartilage is very rich in cells, as it was in Case No. 1; all the islets of cartilage tissue, in both specimens were surrounded by a tough capsule of connective tissue. There is, however, one condition in this case, which was wanting in the former and which may help us in finding out the origin of the cartilage tissue.

In nearly all of the cartilage tissue islets there is a capillary blood-vessel, mostly centrally located and surrounded by a smaller or larger quantity of a tissue, consisting of long, fine, spindle-shaped cells which often have several offshoots, and are embedded in a hyaline matrix, which cannot be stained by carmine. This tissue is closely similar to myxomatous tissue and I think represents nothing, but the remains of the vitreous body thus altered. In the largest, probably oldest islets of cartilage tissue, this myxomatous tissue is wanting or greatly reduced in quantity.

I am therefore inclined to believe that the cartilage tissue takes its origin from the altered remains of the vitreous body. This is the more probable, since the myxomatous tissue gradually passes over into the cartilage tissue, whilst the latter is separated from the sarcomatous elements in every

instance by a capsule of tough connective tissue. During the growth of the cartilage tissue, the myxomatous tissue is destroyed and the blood-vessels become atrophic, and the cartilage tissue islets are then, as I found in Case No. 1, in the following condition, viz., embryonic, growing cartilage in the centre, and stationary cartilage at the periphery.

Perhaps this is not always the manner in which cartilage-tissue is formed in intraocular tumors. I am inclined, however, to apply the same theory in No. 1, although I will by no means deny the possibility of a transformation of the formative sarcoma cells into cartilage, to which the conditions in that case seemed to point.

ON THE THERAPEUTIC USE OF JABORANDI AND MURIATE OF PILOCARPINE IN EYE-DISEASES.

By Dr. M. LANDESBERG, of Philadelphia.

JABORANDI and Pilocarpine have proved in my experience to influence favorably the absorption of intraocular hemorrhages, opacities of the vitreous and aqueous humors in a more reliable and effective manner, than any other absorbent remedy known up to the present time. The unpleasant symptoms, which occasionally follow both the use of Jaborandi and Pilocarpine are only temporary, even in those cases in which the complications forbid further continuance of the remedy. No unfavorable action on the heart, the lungs, nerves, and the general circulation has been observed. The subcutaneous administration of Pilocarpine is simple; it is not followed by any irritation or abscess at the point of injection, and has generally been better borne than the internal administration of Jaborandi. The action of either preparation is different on different persons. The rule must therefore be observed to begin at first with the smallest dose, and to act afterwards according to individual indications. Of course, there are patients who seem to have a positive idiosyncrasy against this remedy, whom the smallest dose prostrates so completely that further administration is impossible.

In the greatest number of cases headache, scintillation before the eyes, retching, nausea and vomiting, pain in abdomen and genitals, followed the use either of Jaborandi or Pilocarpine. Slight gastric catarrh was more or less

common to all patients, and all without exception suffered during the whole course of treatment from great thirst and dryness of the throat.

A *Extractum Jaborandi fluidum* was used in the following cases.

1. *Hemorrhage of the anterior chamber*, filling up the latter to its upper third, with intense subconjunctival injection and ecchymosis of the conjunctiva of the eyeball and of the lids. Vision reduced to quantitative perception of light. This condition had been caused, according to the statement of the patient, a workman 36 years old, by a blow, which he received on the right eye the previous night. 90 drops of fluid extr. jabor. produced profuse perspiration and ptyalism. Secretion of the lachrymal gland was somewhat increased. Slight nausea and tinnitus of the ears were the only secondary symptoms. After six doses, taken in six succeeding evenings, there was an almost total absorption of the blood of the anterior chamber. Vision $\frac{20}{30}$. Vitreous and background of the eyes normal.

Hemorrhage of the anterior chamber and the vitreous, and probably *peripheric detachment of retina*.—Vision: counts only fingers at 2′. Patient, a laborer 56 years old, was struck by a piece of wood on the left eyeball, three days previously to his coming to my notice. Sixty drops of Jaborandi produced a powerful effect, and the after symptoms were very severe. I reduced the dose to 30 and 40 drops respectively, which caused profuse perspiration and salivation; but emesis always occurred, preceded by headache and followed by nausea. After 10 doses, hemorrhage of the anterior chamber and of vitreous had vanished. Vision $\frac{20}{40}$. Field of vision normal. Two small hemorrhages near macula lutea. No detachment of retina.

3. *Hemorrhage of the anterior chamber*, filling up the latter totally. Perception of light and movements of the hand only in centre of F. Patient a bricklayer, 31 years old, was struck by a piece of stone on his right eye, 24 hours previous to my examination. Paracentesis corneæ was followed by hemorrhage and cyclitis.

50, respect. 30 drops of extr. Jabor. fluid. produced such violent symptoms of vomiting, headache and pain in epigastrium, that patient resisted further treatment. Examination five months afterwards showed *concentric rupture of retina around the optic disc with large deposits of pigment* and *atrophy of the papilla*.

4. *Hemorrhage into the anterior chamber and the vitreous.*—Only quantitative perception of light, which is doubtful upwards. Patient, a laborer's wife, 50 years old, received a blow on her left eye from a horse's hoof two weeks previously to her coming to my notice. 80, respect. 100 drops of extr. Jabor. fluid. produced profuse perspiration and salivation. After symptoms slight. After 14 doses the blood in vitreous was totally absorbed. There was slight detachment of retina in its inferior part and a large apoplexy around the optic disc, partly covering the latter. Vision: counts fingers at 2'.

5. *Opacities of vitreous of both eyes consequent upon serous Iridochoroiditis.*—Vision $\frac{20}{40}$. Patient, a barber's wife, 27 years old, was put seven times under the influence of extr. Jabor. fluid., 50 drops of which caused profuse perspiration. The after symptoms as vomiting, scintillation before the eyes, headache and pain in abdomen and genitals, were very violent. Result: perfect absorption of the opacities of vitreous. Vision of the right eye, $\frac{20}{20}$, of the left eye, $\frac{20}{30}$. Jaeg. 1 with either eye.

6. *Intermittent, alternating Mydriasis* in connection with irregularities of the menstrual function. Patient, a healthy girl, 17 years old, states that for two days she had suffered from intermittent, alternating dilatation of her pupils, which lasted only a short time, entirely subsided to reappear, after a short free interval, now in the right, then in the left eye.

Menstruation, having been irregular from the beginning, had stopped for five months. At regular periods of three weeks patient suffers from flushing of the face, slight pain in the regions of the womb and the spine, which is followed and relieved by bleeding from the nose. I advised patient to take 50 drops of extr. Jabor. fluid. the day the first symptoms of her suppressed period were felt, and to repeat the dose for three days. The remedy was very well borne; perspiration was profuse; menstruation reappeared, becoming henceforth normal; intermittent mydriasis subsided entirely.

7. *Atrophy of papilla nervi optici of both eyes.—Right eye:* Counts fingers at 15'. Reads without glasses Jaeg. 10; with +10 Jaeg. 8. *Left eye:* Counts fingers at 18'. Reads without glasses Jaeg. 7; with +10 Jaeg 5. Central scotomota of both eyes. Field of vision of both eyes is limited upwards and inwards. The line of limitation is lower in the right eye. Patient, a barkeeper, 33 years old, had suffered from syphilis previously to the

beginning of his eye-trouble, under which he has been laboring for five years. He had been treated with large doses of mercury and iodide of potassium. He had been largely addicted to liquor and had had attacks of delirium tremens. The treatment with extr. Jabor. fluid. in doses of 80 drops, which did not produce any unfavorable after-symptoms, had the following result.

1. *After the 8th dose:—R. E.*—V. $\frac{15}{200}$. Jaeg. 8 without glasses; w +10 Jaeg. 6. *L. E.*—V. $\frac{20}{200}$. Jaeg. 5 without glasses; with +10 Jaeg. 4.

2. *After the 16th dose:—R. E.*—V. $\frac{20}{100}$. Jaeg. 5 without glasses; with +10 Jaeg. 3. *L. E.*—V. $\frac{20}{70}$. Jaeg. 4 without glasses; with +10 Jaeg. 2.

3. *After the 23d dose:*—Vision of each eye $\frac{20}{70}$. Each eye reads without glasses Jaeg. 3 fluently. Jaeg. 2 with some trouble. With +10 Jaeg. 1. Field of vision much enlarged. Central scotomata entirely subsided. Color of the optic discs reddish. Veins normal; arteries more filled than at the beginning of the treatment.

B. *Muriate of Pilocarpine was used in the following cases:*

1. *Choroiditis absoluta peripherica with circumscribed choroidal atrophies and pigment infiltrations into the retina of both eyes. Large floating opacities of vitreous in the right eye, smaller ones in the left eye.*—Vision of the right eye $\frac{1}{30}$. With +10 Jaeg. 12. Vision of the left eye $\frac{1}{20}$. With +10 Jaeg. 5. Duration of the affection of the right eye for eight months, of the left eye for some weeks. Patient had been treated with large doses of iodide of potassium. An 18 days treatment with subcutaneous injections of Muriate of Pilocarpine in doses of one quarter of a grain, at first every day, then every other day, had the following result:—*Right Eye:*—Only very few and thin opacities of vitreous remaining. V. $\frac{1}{30}$. With +10 Jaeg. 3.

Left Eye:—Opacities entirely vanished. V. $\frac{1}{15}$. With +10 Jaeg. 1. Patient, merchant's widow, 60 years old, suffered in the course of the injections from asthma four times, from vomiting five times, from abdominal pain once. Perspiration and ptyalism were very profuse.

2. *Intraocular hemorrhages occurring after Sclerotomy*, made on the right eye of a musician, 43 years old, who suffered from Glaucoma absolutum with acute exacerbations. Sclerotomy was followed by very severe symptoms of irritation, violent ciliary neuralgia and repeated hemorrhages in the anterior chamber and

vitreous. Paracentesis only aggravated the condition. Eserine was of no avail. Intraocular pressure increased, the lips of the wound were forced apart and prolapse of iris occurred.

Iridectomy stayed the morbid process, but not the slightest absorption of the hemorrhage took place. In this condition I absorpted to subcutaneous injections of one-half of a grain of Pilocarpine pro dosi, in order to favor absorption.

After five injections hemorrhage and chemosis had almost totally disappeared. Intraocular pressure was diminished. Vomiting occurred only once. Slight asthma and rushing in the head were the only unpleasant after-symptoms.

3. *Opacities of the vitreous consequent upon Irido-choroiditis with anterior Synechiæ of both eyes.*—The affection was chronic. Patient, a baker, 36 years old, had been treated by me with mercury and iridectomy, which brought vision of the right eye from $\frac{2}{10}$ and Jaeg. 8 to $\frac{3}{10}$ and Jaeg. 2, and vision of the left eye from $\frac{2}{100}$ and Jaeg. 13 to $\frac{2}{10}$ and Jaeg. 5. Opacities of vitreous remained unchanged. To induce absorption I resorted to subcutaneous injections of Pilocarpine in doses of one-third, resp. one-quarter of a grain. Perspiration and ptyalism were very profuse, the unfavorable after-symptoms very severe. After each injection patient suffered from asthma, pressure in epigastrium and emesis. Eight injections brought the following result: *R. E.*—V. $\frac{3}{10}$, Jaeg. 1. Vitreous perfectly clear. *L. E.*—V. $\frac{3}{10}$, Jaeg. 2. A few very fine opacities.

4. *Choroiditis acquatorialis absoluta with circumscribed choroideal atrophies, pigment infiltration into the retina and floating opacities in vitreous. Cataracta incipiens of both eyes.*—V. *R.* $\frac{1}{10}$. V. *L.* $\frac{1}{10}$. With +10 *R. E.*, Jaeg. 5. *L. E.*, Jaeg. 8. In order to induce absorption of the opacities of vitreous, and to give a better chance for a future operation for cataract, I proposed a treatment with subcutaneous injections of Pilocarpine, to which patient, a carpenter's wife, 50 years old, readily submitted. On one-quarter of a grain of Pilocarpine the after symptoms were most violent. Fever observed. Vomiting, asthma, pain in abdomen and genitals continued for two hours. General prostration and nausea kept on during the following twelve hours. When, after a pause of three days, the injection of one-sixth of a grain of Pilocarpine was followed by the same unfavorable symptoms, I restrained from further trials.

5. *Aequatorial choroiditis. Serous infiltration of papilla and*

Retina. Membranous opacities of vitreous. Intense inflammatory symptoms. Iritis with posterior Synechiæ.—Vision, $\frac{5}{40}$, but only by uplifting the eye to the utmost; with central fixation: Vision only $\frac{5}{100}$ and Jaeg. 19. Patient, a farmer, 25 years old, had suffered from serous irido-choroido-retinitis of the same (left) eye, and had made a complete recovery consequent upon a mercurial treatment combined with the use of decoctum Zittmanii. The acute mercurialization failed to check the progress of the morbid process, which brought about *detachment of choroid*, downwards from the optic disc, at the distance of a papilla-diameter. Subcutaneous injections of Pilocarpine in doses of half a grain seemed at first to improve the condition of the eye; but after the fifth administration relapse occurred. There was: detachment of choroid and retina (?) in the whole inferior half, phthisis of the eyeball, and vision reduced to counting fingers at 2′. After three more injections, which did not afford any benefit whatever, the treatment was given up. The injections were well borne. Slight nausea and scintillation before the eyes were the only after-symptoms.

6. *Detachment of retina in its whole inferior half*, of eight months standing.—Vision: counts fingers at 8′ with +10. Jaeg. 18 with +5. Field of vision cannot be measured. Great torpor retinæ. Perception of colors impaired. Ultramarine appears to be now green, then blue. Violet, Indigo-blue and Prussian-blue appear dark. Patient, a merchant's wife, 31 years old, descends from a family in which myopia is hereditary. She has always been near-sighted, and myopia rapidly increased in the last years, due to over-exertion of the eyes. In April of 1878, *detachment of retina of the left eye* occurred. She had been under treatment and kept in bed in a dark room for eight days—without any benefit. Subcutaneous injections of Pilocarpine had the following effect:

1. *After the fourth injection:*—V. $\frac{5}{60}$ with +12. With +5 some words of Jaeg. 12. Retina re-attached in all parts with the exception of the inferior inner quadrant. Field of vision much enlarged. Central fixation restituted.

2. *After the eighth injection:*—V. $\frac{5}{60}$ respecl. $\frac{5}{40}$ with +14. With +5 Jaeg. 6. Detachment of retina of the inferior-inner quadrant much smaller.

3. *After the 17th injection*, at the end of the treatment:—V. $\frac{5}{60}$ with +18; without glasses, V. $\frac{5}{100}$. With +10 some words of Jaeg. 6 and 5. There is only a small flat detachment in the in-

ferior-inner quadrant of the retina. Field of vision limited only upwards and inwards. Slight degree of torpor retinæ. Perception of colors: Yellow appears white; light-blue, Prussian-blue appear green; ultramarine and Indigo-blue appear dark-blue.

Four weeks later the examination showed: V. $\frac{8}{40}$ with +18. With +10 some words of Jaeg. 5. No change in the condition of the retina. In the course of the administration of Pilocarpine, which had an energetic diaphoretic and sialogogue effect, vomiting occurred three times. A slight degree of nausea took place after each injection. Slight gastric catarrh set in after the eighth injection, which kept on during the whole treatment.

7. *Total detachment of retina in its inferior half and large floating opacities of vitreous.*—Affection of the right eye occurred consequent upon a violent fit of coughing. No history of myopia. Patient, a widow, 66 years old. Beginning of the treatment with Pilocarpine eight weeks after the occurrence. Six injections of one-third of a grain did not effect any change whatever in the condition of the detachment of retina. Vomiting occurred only once. Nausea and uneasiness were slight, perspiration and ptyalism profuse.

8. *Retinitis e morbo Brightii in both eyes. Large diffuse apoplexies of the right eye; striated hemorrhages in the circum-papillary region and some hemorrhagic patches along the vessels of the left eye.* —*Right Eye:*—Hardly quantitative perception of light. Pupil moderately dilated.

Left Eye:—V. $\frac{1}{10}$. With +10 Faeg. 8. Pupil somewhat dilated and sluggish. Field of vision and ocular tension normal. Patient, a liquor dealer, 57 years old, largely addicted to intemperance, has suffered for two years from Bright's disease. There is slight ascites, swelling of the legs and hypertrophy of the left ventricle. He had never suffered from uræmic attacks and had never been subject to headache, convulsions or spasms. There is no history of syphilis. Impairment of vision had set in five months previously to his coming to my notice. My trials with hypodermic injections of Pilocarpine in daily doses of one-third of a grain had the following result: 1. *After the sixth injection:*—V. *L.* $\frac{1}{10}$. With +10 Jaeg. 6. General condition much improved. Dropsy and amount of albumen in urine diminished. 2. *After the tenth injection:*—V. *L.* $\frac{1}{10}$. With +10 Jaeg. 4. Pupil of normal dilatation and reaction. Some hemorrhagic spots in the retina are absorbed. Optic disc is less choked. No

improvement whatever of the right eye. The administration of Pilocarpine was very well borne. Perspiration and ptyalism were very profuse. Nausea only inconsiderable, and not the slightest unfavorable effect on the action of the heart.

9. *Atrophia nervi optici absoluta. Amaurosis of the right eye. Neuritis optica of the left eye*:—V. $\frac{20}{100}$ Jaeg. 11. With +10 Jaeg. 8. Field of vision limited upwards and inwards. Reaction of pupil very slow. Anamnesis very uncertain. Patient, baker, 42 years old, ascertains that he had always been subject to violent headache, and that for two years he has suffered from sleeplessness. He cannot state the beginning of his eye-affection. The right eye blind five months. No syphilitic history. No suspicion of tabes dorsualis or diabetes mellitus. A mercurial treatment combined with Heurteloups brought vision of the left eye to $\frac{20}{40}$ and Jaeg. 5 with +10. The inflammatory symptoms of the optic disc decreased. Pupil reacted more promptly on light. No improvement of the field of vision. I put patient under the influence of subcutaneous injections of Pilocarpine in daily doses of one-third of a grain, which gave the following result: 1. *After the sixth injection*:—V. *L.* $\frac{20}{60}$. With +10 Jaeg. 3. 2. *After the twelfth injection*:—V. *L.* $\frac{20}{30}$. With +10 Jaeg. 2. Reaction of the pupil normal. All inflammatory symptoms of the background of the eye vanished. Optic disc somewhat flat, of reddish color. Veins normal, arteries somewhat thin. 3. *After the eighteenth injection*:—No change in the condition of vision and of the background of the eye. Field of vision remains contracted upwards and inwards. General health of the patient much improved. He is now able to sleep for some hours during the night, and has been free from headache for about two weeks.

The antagonism between Pilocarpine and atropia I observed in the case of Mrs. B., suffering from detachment of retina of the left eye.

Her right eye had been kept under the influence of atropia for some weeks previous to the beginning of the Pilocarpine-treatment. I administered at first one-sixteenth of a grain, which had not the slightest effect. When the dose had reached one-half of a grain, ptyalism set in, but no trace of perspiration. The latter was very slight from three-quarters of a grain, and increased somewhat when the dose reached one grain. The face was cool and damp, the body hardly warm.

From the third injection, the effect of half a grain of Pilocar-

pine was very prompt, nothwithstanding the instillation of atropia was continued.

In regard to the myotic qualities, I must claim the great superiority of Eserine to Pilocarpine. The former has proved in my experiments to be a more powerful and more efficacious remedy than Pilocarpine, and its use is attended either by very slight or no inconvenience to the patient, whereas Pilocarpine possesses the very disagreeable properties of increasing the action of the lachrymal gland and producing ptyalism, if absorbed by the blood.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY FOR THE FIRST HALF OF THE YEAR 1878.

By J. HIRSCHBERG, of Berlin.
Translated by Dr. R. O. Born, of New York.

The following report will be concise, but will contain everything of importance, so as to make the Archives a real organ of ophthalmology, which shall furnish a true mirror of the present *status* of ophthalmic science and art. If we compare these Archives with periodicals of the same kind and extent, we see that besides original papers, reports are now given by the *Annales d' Oculistique*, the *Annali di Ottalmologia* and the *Ophthalmic Hospital Reports*. Von Graefe also had the same intention with his *Archives*, (preface to 1, 2, iv., 2.), but was unable to carry it into effect. His words may enter into our programme: "By giving a comprehensive and critical review of the whole German and foreign literature, we hope to be of service to a large number of the profession, the more so, as we do not intend to present disconnected extracts, but a complete and systematic résumé."

I.—Anatomy and Physiology.

1. *Intercellular lacunæ of the anterior corneal epithelium* were found by Th. Leber of Göttingen. (*A. f. O.*, Vol. XXIV, 1, pag. 252–294), in inflammatory processes, where during life the corneal surface presented the well-known *dull, lustreless appearance.*

2. In the rabbit the strongly refracting basal margin at the lower end of the deepest layer of epithelial cells may be mistaken for an anterior elastic membrane of the cornea, (Stirrling and Skinner, J. of physiol., Vol. 1, 4 and 5, pag. 335).

3. According to Dr. Senftleben, (*Virchow's Arch.*, Vol. LXXII, pag. 542), the *pure keratitis* depends exclusively upon the *migrating cells*, which enter the exposed corneal tissue either from

the periphery of the cornea (marginal loops), or from the conjunctival sac.

4. Researches on the microscopical structure of the cornea, by HASSLOCH of New York, (*A. O. & O.*, Vol. VII, 1, pag. 3–11).

5. W. GOLZIEHER of Buda-Pest, (*C. f. A.*, 1878, pag. 2 and ff, with illustration), examined an eyeball with *pterygium* enucleated from the dead body of an old woman, and confirms Arlt's view that pterygium is a duplicature of the conjunctiva which by cicatrization is drawn over the cornea to cover defects of that membrane.

6. A *communication of the anterior chamber* with the anterior ciliary veins is stated by F. HEISRATH, (*A. f. microsc. Anat.*, Vol. XV, 2, pag. 209), after injections into the eyes of living animals.

7. ARNSTEIN of Kasan, (*C. f. m. W.*, 1878, No. 7), found in the iris of the albinotic rabbit three different *terminations of nerves:* 1, a terminal network of motor nerves in the sphincter; 2, of sensitive nerves near the anterior surface of the iris; 3, of vasomotor nerves around the arteries and capillaries.

8. The layer of radial fibres in the *lens of birds* by K. RITTER, (*A. J. O.*, Vol. XXIV, 2, pag. 1–3, with illustration).

9. *Outer segments of rods* in the retina of the frog by DIETL of Innsbruck. (*A. O. & O.*, VII, 121).

10. *Disease of the retina in leucocythæmia* by DEUTSCHMANN of Göttingen, (*Z. M.*, May, 1878). Numerous hæmorrhagic patches in the retina without a white centre; mostly at some distance from the bloodvessels. Moreover, accumulations of white blood corpuscles surrounded by a zone of red ones. Finally a purely white patch consisting of sclerosed nerve fibres. Vessels distended, filled with blood corpuscles, especially white ones. The same in the choroid. Odema of the optic discs.

11. Contributions to the pathological anatomy of the eye in LEUCOCYTHÆMIA by Pettel, (*A. f. O.*, Vol. XXIV, 3).

12. Atlas of the *pathological topography* of the eye by O. BECKER of Heidelberg, 3d part. Vienna, 1877, Braumüller. (Treats especially of Cyclitis, Irido cyclitis, etc., with most excellent illustrations and short descriptions; also intraocular cysticercus, foreign bodies in the anterior of the eye, etc.).

13. Contributions to the pathological anatomy of the eye by SCHOTT of Innsbruck, (*A. O. & O.*, Vol. VII, pag. 159–178).

14. A case of bilateral congenital *Anophthalmia* by WICHERKIEWICZ of Posen, (*Z. M.*, April, 1878). Eyeballs absent, annexa developed, etiology entirely obscure.

15. A case of bilateral anopthalmia by J. MICHEL of Erlangen, (*A. f. O.*, XXIV, 2, pag. 71–83, with illustrations). In a deformed newborn child the orbits were small and contained only a body, the size of a pea, made up by cartilage and tissue of the lachryneal gland and muscle, and besides a conjunctival sac *partly* closed by growing together of the lids.

16. *Coloboma of the Iris and Choroid* by H. PAUSE of Erlangen, (*A. f. O.*, XXIV, 2, pag. 84–92, with illustrations).

17. Contributions to the congenital malformations of the eye by O. HAAB of Zurich, (*A. f. O.*, Vol. XXIV, 2, pag. 257–284, with illustrations).

PART I will contain the *normal and Pathological Anatomy and Physiology.*
PART II the *Therapeutics.*
PART III the *Special Pathology.*
PART IV the *other Literature of Ophthalmology.*

The next number of the Archives will conclude this period; the literature of the second series of 1878 will be reviewed in a similar manner in the third and fourth numbers. In the interest of science and in their own, authors are respectfully requested to send us reprints, reports and books.

ABBREVIATIONS.

A. d'O . .—*Annales d'Oculistique*, continuées par Warlomont, Bruxelles.
A. d'Ott. . .—*Annali d'Ottalmologia* direi dal prof. Quaglino, Milano, Vallardi. (7th year.)
A. f. O. . .—*Graefe's Archiv f. Ophthalm.*, edited by Arlt, Donders and Leber. Berlin, H. Peters. (24th year.)
C. f. A. . .—*Centralbl. f. prakt. Augenhlkde*, edited by J. Hirschberg. Leipsic, Veit & Co.
Cr. O. . . .—*La Cronica Oftalmologica*, Dir. Dr. D. Cayetano del Toro, Cadiz. (Galvez.)
J. d'O. . .—*Journal d'Oculistique et de chirurgie*, edited by Fano of Paris.
A. O. O. . .—*Archives of Ophthalmology and Otology*, edited by Knapp, Moos and Mauthner, 7 volumes. Papers published in this journal are mentioned only by titles.
O. H. R. . .—*Ophthalmic Hospital Reports*, edited by Jonathan Hutchinson, London, Churchill. In irregular numbers, 9 volumes.
R. d'O. . .—*Recueil d'Ophthalmogie*, edited by Galezowski and others, in Paris.
Z. M. . . .—*Klinische Monatsbl. f. Augenheilkunde* edited by W. Zehender, Cassel, Th. Fischer.

To these Journals of Ophthalmology have been added during 1878 *:*

P. O. . . .—*Periodico de Ophth. pratica*, by Van der Laan and Fonseca, Jr., at Lisbon.
G. O. . . .—*Giornali delle malattie degli Occhi p. dal* Dr. F. Morano, Naples.

E. . . . .—emmetropia, e—emmetropic.
M. . . . .—myopia, m—myopic.
H. . . . .—hypermetropia, h—hypermetropic.
As. . . . .—astigmatism, as—astigmatic.
A. . . . .—range of accommodation.
V. F. or F. .—visual field.
On. . . . .—ophthalmoscopically normal, n—normal.
V. or S. . .—acuteness of vision.
Vc. . . . .—acuteness of central vision.
Ve. . . . .—acuteness of eccentric vision.
μ. . . . .—index of refraction.
λ. . . . .—length of axis.
ϱ. . . . .—radius of curvature of the cornea.
P. . . . .—diameter of the optic disc (about 1,5 mm.)
r. . . . .—right, right-sided.
l. . . . .—left, left-sided.
y. . . . .—year, years.
T. . . . .—tension.

COLOR-BLINDNESS.

18. *De la cécité des couleurs* dans ses rapports avec les chemins de fer et la marine par F. HOLMGREN, Prof, d. Physiol. à l'Un. d'Upsal, Stockholm, year not stated ; and Paris, Masson,* Paris.

A railroad accident aroused the suspicions in HOLMGREN that color-blindness might have been the cause of the accident, and led him to his researches. Among 2200 soldiers he found 12 red, 17 green, 1 violet blind, and 31 incompletely blind,† *i. e.*, 2.7 per cent. with wrong perception of colors. Among 266 railroad officials 4.8 per cent. with deficient sense of color, viz. : 6 G. B., 7 I. B. KROHN found among the 1200 railroad officers of Finnland, 5 per cent. with imperfect perception of colors, 4 R. B., 25 G. B., 31 I. B.

Following the *Young-Helmholtz* hypothesis (of three subjective principal colors, red, green, and violet), Holmgren distinguishes.

I. *Total color-blindness ;* II. *Partial color-blindness.* (*a*) Blindness for red, (*b.*) for green, (*c*) for violet. 2. *incomplete color-blindness* which through a weak sense of color passes over the normal. Since in railroad and marine service red signals indicate danger green ones security, color blind persons should be excluded from the service.

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* Published in German by Vogel of Leipzig, 1878.

† R. B., Red-blind. G. B., Green-blind. V. B., Violet-blind. I. B., Incompletely color-blind.

Holmgren's expeditious and certain test of color-blindness with wool,* is as follows: I. A light-green piece of wool is put aside from the others. The person to be examined has to add those of the same color. The examination is continued until all the samples of the same shade have been added or besides them also those of a grey, reddish, rosy color. II. A purple † sample is put aside. The person who had by I been recognized as color-blind, has incomplete color-blindness, if he adds only purple shades. Those who add besides them blue or violet or both are entirely red-blind. Whoever besides the purple adds green or grey or both, is entirely green-blind. Those who besides the purple add red or orange, are violet respectively blue-blind. III. Also a red sample may be put aside. The red-blind add green or brown shades, which appear darker to the healthy eye than the red of the sample. The green-blind will add such shades of green or brown which appear lighter to the normal eye.

19. A simpler method was published by HOLMGREN in *C. f. A.* 1878. pag. 177–187.

20. Dangers from *color-blindness* in railroad employés and pilots, by JOY JEFFRIES, M. D., of Boston. B. Rand, Avery & Co., 1878. In America among 8,861 whites 2 per cent. were found color-blind. The examination was not very accurate. The book contains a general description of the subject, and a very complete record of the *literature.*

21. Lecture on color-blindness by the same. *Boston Daily Advertiser.*

22. Incurability of color-blindness by the same.

23. On *color-blindness* (*Breslauer Zeitung*, No. 59, 1878. *C. f. A.*, 1878, pag. 56 ff). At the meeting of the section for public hygiene, on February 1st, 1878, MAGNUS, H. COHN, and JACOBI spoke on color-blindness. Magnus relates the historical development of the sense of color. Cohn speaks about the nature of color-blindness, and the different methods of examining it with pigment colors, *especially with the wool-samples of Hohngren*; with contrast colors; with spectral colors, especially with the spectroscope constructed after *Vierordt* by *Schmidt* and *Haensch* in Berlin, which by a simple slide allows the presentation of any spectral color alone). A preliminary communication is given of the examination for color-blindness of many school-children of Breslau, (see

* A collection of the woolen samples can be obtained from Frl. L. Oldberg, in Upsala, and from P. Dörffel in Berlin, for five mark.

† Purple is red and blue, or red and violet. Light purple, rosa.

below). Finally Jacobi discusses the hygienic importance of this examination.

24. Further remarks on the theory and practice of color-blindness, by MAGNUS of Breslau. (*Deutsche Med. Wochenschr.*, 1878, No. 20.)

Among fortyseven color-blind persons two were blind for all colors. Magnus recommends Hohngren's method on the strength of his own experience.

25. The color-blindness, its nature and importance. Presented to magistrates, physicians, teachers, etc., by Dr. H. MAGNUS, Lecturer on Ophthalmology at the university of Berlin, 1878. Breslau, published by J. U. Kern, 64 pages.

26. Contributions to the knowledge of color-blindness. Inaugural-dissertation (under the auspices of Prof PFLÜGER), by Dr. F. MINDER. Berne, 1878, *Stämpfle*. General remarks. Holmgren's method, Rose's chromatometer and Meyer's contrast experiment are recommended.

27. *A. Daae* of Kragerœ, Norway (*C. f. A.*, 1878, pag. 10, and separately published by *P. Dörffel* of Berlin, with the wool-samples) has edited a plate of wool-samples for the detection of color-blindness, with explanatory remarks. The plate contains ten horizontal colored rows, and each row seven colors (wool-threads). A. Three rows are *isochromatic* with different shades (3 purple, 7 green, 6 red). B. The other rows are *pseudo-isochromatic, i. e.*, they contain colors which may appear alike to the color-blind, but never to the healthy. If the examined person pronounces the colors of one of the rows under B as being the same color, he is color-blind.

28. A new edition has been published during the summer of 1878, by P. Dörffel and A. Hirschwald with new explanations and wool-samples. This considers the blue-yellow-blindness.

29. Embroidered letters (for example light-blue on pink background, etc., made from wool), were used for the detection of color-blindness by H. COHN, (*C. f. A.*, 1878, pag. 77), since STILLING's pseudo-isochromatic plates did not prove satisfactory to him. The wool used for the letters must be as thick as that for the background ; the letter must neither be raised nor depressed. It is advisable to have the letters pressed, when embroidered.*

30. *Plate for testing and exercising* the sense of color, by J. HIRRLINGER, painter at Stuttgart, Stuttg., 1878, *Paul Moser*.

* COHN will have his samples copied and published.

31. Dor has *empirically* stated the sizes of colored round discs which can be recognized by a normal eye at from 5 to 20 meters distance, (with daylight and in the dark chamber, the normal candle being 20 *cm.* distant from the blackboard), and has constructed plates for the numerical determination of the sense of color, which he uses in combination with *Donders'* apparatus of colored glasses. (Echelle pour mesurer l'acuité de la vision chromatique. Paris, *Masson*, 1878).

32. Compare also Dor and Favre. Nouvelles recherches sur la déterm. quant. de la vision chromatique Lyon, Assoc. typogr., 1878.

33. A. Weber of Darmstadt has made empirical test objects, (colored paper on velvet background), for the distance of $\frac{1}{20}$ *m.* (*C. f. A.*, 1878, Sep., XXVIII. Published by *Ehrhardt* and *Metzger* at Darmstadt, Elisabeth st. 47).

34. Stilling has *re-edited* his *pseudo-isochromatic plates*, (first for red-green blindness) with introduction. The plates do not show the least lustre any more, and may therefore be used without precautions also by the inexperienced. (The examination of the sense of color of the railroad and marine employés, by Dr. *J. Stilling*. Cassel, 1878, Th. Fisher. 1).

35. H. Cohn of Breslau, (*C. f. A.*, 1878, pag. 35), employs, after the old experiment of *Ragona-Scina*,* the *simultaneous contrast for the diagnosis of color-blindness*. In bright daylight we take a strip of thick white canton paper which is folded in the middle at right angles, and a blot of ink (or a piece of black velvet) on both inner halves, and some colored glasses. If the right angle is divided into halves by a red glass-plate, and if we look at the glass from above, the one spot appears red by reflection, the other bluish-green, etc., by refraction. Color-blind persons do not perceive the contrast colors, but call the green spot, dark blackish or blueish †.

36. Pflüger of Berne, (*C. f. A.*, 1878, pag. 50), has used the same method for three years and finds it convenient, where many persons have to be rather accurately and expediously examined for their sense of color. He also uses, according to *A. Weber*, *Meyer's* contrast experiment in *v. Bezold's* modification: black print on colored ground, covered with transparent (tissue)-paper,

* 1847. Conf. *Helmholtz*, Physiol. Opt., pag. 405; *v. Bezold*, Farbenlehre pag. 183; *Brücke*, Physiol., Bd. II., pag. 141; *Aubert*, Phys. Opt., in *Graefe-Saemisch*, Bd. II., pag. 548.

† The apparatus has plates of metal and can be folded; sold by *P. Dörffel* of Berlin.

appears in the complementary color, [black print on red ground appears light-green, on yellow ground blue, on green ground purple, on blue ground yellowish-brown, on violet ground yellowish-green].

37. *Colored shadows in daylight*, by J. STILLING of Cassel, (*C. f. A.*, 1878, pag. 124). If a colored plate of glass is inclined with one end toward the window under an angle of 45°, and the other end rests upon a sheath of white paper, a stripe appears upon the latter near the glass, which shows the complementary color of the glass.

38. H. COHN, (No. 33), demonstrates the contrasts of the colored shadows by a special chromaskiopticon.

39. F. HOLMGREN, (Upsala, Läkaref. Förhandl., XIII and *C. f. A.*, 1878, pag. 213), has constructed a special instrument, (chromato-skia meter), for the observation of colored shadows.

40. MAGNUS of Breslau, (*C. f. A.*, 1878, pag. 80), lets, in the spectroscopic examination for color-blindness, the patients pick out those wool samples (of *Holmgren*), which appear identical to the color seen in the spectroscope and thus makes them reproduce the entire spectrum, and also the Natrium, Lithium and Thallium lines.

41. H. COHN has used a similar (nerve extensive) method. (*Bericht über die Heidelberger Vers.*, 1878, conf. also *C. f. A.* Oct., Nov. and Dec.).

42. J. HIRSCHBERG has (by *P. Dörffel*), *Vierordt's* spectroscope modified in such a manner, that every stripe of the spectrum can be directly compared with the others, (*C. f. A.*, Oct., 1878), in order to decide whether *color-blind persons really confound spectral colors.* (This is indeed the case. The instrument works beautifully).

43. WEINHOLD, (*Poggendorff's Annals*, 1877, No. 2, pag. 631–658), distinguishes—against *Hering*—the red- from green-blindness. The red-blind confounds light-red with dark-green, the green-blind light-green with dark-red. A green-blind person (to whom green, light-purple and gray appear alike), perceives the spectrum up to its normal limits, between E. and 1, he sees a gray zone. For a red-blind patient the red end of a spectrum was a little shortened, the less refracting half was called yellow, the other blue. The colorless limit was situated in blueish-green.

44. *In blue-yellow-blindness* the spectrum is mostly, but *not always* (5:1) shortened at the refracting end. (J. STILLING of Cassel, *C. f. A.*, 1878, pag. 99).

45. The color-blind DELBOEUF, (*D.* and *Spring*, Bull. de l' Ac. r. de Belg., Bd. XLV, No. 1, 1878), could not distinguish directly a red from a brown *ribbon* on the one hand, and a violet from a blue one on the other, but very well through a solution of fuchsine which casts a shadow especially over the green portion of the spectrum. Red-blindness is therefore interpreted by the authors as an *excessive sensitiveness for the violet and green rays.* Through chloride of nickel, (which transmits especially green), red and brown on one hand, and blue and violet on the other, may appear alike to a healthy eye; and if we now interpose a layer of fuchsine those colors can again be distinguished.

46. J. HIRSCHBERG of Berlin, (*Verh. d. physiol. G. z.* Berlin, May 31, 1878, also in *E. du Bois-Reymond's* Arch. f. Physiol.) The ordinary form of so-called "color-blindness" is *green-amblyopia.* The spectrum has its normal expansion, and consists of "*yellow and blue*" with a colorless (gray) interposed stripe, corresponding to the pure spectral green; *the lightest portion of the spectrum lies in the yellow*, as in the normal eye.

47. Of 1,312 sailors, 63 or 4.8 per cent. showed deficient sense of color, with *Stilling's* plates. Many persons, however, could not *name the colors correctly.* (*Lederer*, Wiener med. W., No. 2 etc. pag. 1878.)

48. DAAE of Kragerœ, Norway, (*Magazin for Lægeridenskaben*, Bd. VII, pag. 7; and German, *C. f. A.*, 1878, pag. 79), has found among 205 school boys 4.8 per cent. color-blind, (and 5.36 per cent. with weakened sense of color), among 208 girls none color-blind, (only 5 per cent. with impaired sense of color).

49. H. COHN and MAGNUS, (*C. f. A.*, 1878, pag. 97), have examined 5,079 school-children for color-blindness at Breslau. The preliminary examinations were made after *Holmgren* with pink wool, only such were entered into the statistics as proved undoubtedly color-blind, after an examination of several hours, with pigment contrast, and spectral colors. Of 2,761 boys 76 or 2.7 per cent. were color-blind; of 2,318 girls 1=0.04 per cent.

50. F. HOLMGREN of Upsala, (*C. f. A.*, 1878, pag. 202), found among 32,165 men 250 entirely red-blind, 276 entirely green-blind, 493 incompletely color-blind, in all 1,019 or 3.25 per cent. color-blind; among 7,119 females, relatively 2, 1, 16, in toto 19 or 0.26 per cent. color-blind.

51. H. COHN of Breslau, spoke at the Ophthalmological Congress at Heidelberg, 1878, (*C. f. A.*, Sept., pag. 29), of the

observations on 100 *color-blind persons.* Among 2,429 schoolboys he found 95 or 4 per cent. color-blind, among 1,061 girls 0. The best and quickest method of a preliminary examination is with pink wool. 85 were red-green blind, 5 blue-yellow blind, 7 entirely color-blind. *Stilling's* plates require too much, *Daae's* are excellent, also *Snellen's* colored letters.

52. MAGNUS of Breslau, (*ibid.*, pag. 38), found among 2,216 girls 1 color-blind, among 3,273 boys 100 color-blind, *i. e.*, 3.27 per cent.

53. MINDER, (No. 26), found at Berne among 2,275 individuals, 106 (males 6.58 per cent., females 1.3 per cent.) color-blind. 36 G.-B., 15 R.-B., 1 V.-B., 55 I.-B.

HEMIANOPSIA.

54. On hemianopsia* by J. HIRSCHBERG, Contributions to practical ophthalmology, III, 1878, Leipzig, Veit & Co., pag. 1–18, with 5 figures. The hypothesis of a partial crossing of the optic nerve fibres in the chiasma of man, is in harmony with comparative anatomy; with normal anatomy and histology (*Gudden*); and with pathological anatomy, since in typical hemianopsia of the same side in both eyes, the seat of the disease has always been found in the opposite half of the brain (*Hirschberg, Jackson, Förster, Pooley, Jastrowitz*), and since in one-sided amaurosis of an eyeball by a peripheral cause *both* tracts atrophy (*Woinow, Schmidt-Rimpler*); with the experiments on dogs (*Gudden*); and finally with clinical observation. Decisive evidence is furnished by the *mathematical congruity of the separating line in the visual field of both eyes* in cases where the hemianopsia is not total but involves for example, only $\frac{3}{8}$ of each V.-F.

55. *Left bilateral hemianopsia*, (S=1) apoplectic cyst, the size of a walnut, in the *right occipital lobe.* BAUMGARTEN of Königsberg. (*C. f. m. W.*, 1878.)

56. HOSCA of Basel (*Z. M.*, January, 1878) found in a man, 54 years of age, after apoplexy, absence of the *left* half of the visual field of either eye, S almost 1, the papilla reddish. Repeated apoplexies. All the older lesions were found in the region of the right visual centre (Thalamus opt.).

57. Finally, BAUMGARTEN of Königsberg, (*C. f. med. W.* 1878, No. 31), demonstrated *microscopically* in a case, in which v. Hippel

*The name hemianopsia avoids all ambiguity. The defect of the temporal half of the visual field of each eye is called by some authors temporal hemiopia by others more correctly nasal hemiopia; a temporal hemianopsia is self-understood on account of the *a privativum.*

had enucleated *one* eyeball seven years previously, a partial atrophy in *both* optic tracts, and thus subtantiated a very important desideratum for the theory of semi-decussation.

58. H. ULUNK of Berlin, by the extirpation of the cortex of the occipital lobe on one side, in an ape, produced bilateral hemianopsia of the other side. *Verhandl. d. physiolog. G. zu* Berlin, 1878.

59. If in young cats the chiasm is cut (from the mouth) in the sagittal plane, sight is preserved. (*Partial decussation.*) NICATI of Marseille (*C. f. m. W.*, 1878, No. 25).

60. GOWERS of London, (*C. f. m. W.*, 1878, No. 38), found *a* in atrophy of *one* optic nerve, increase of connective tissue in *both* optic tracts ; *b*, in *left-sided* hemianopsia and hemiplegia a tumor which had destroyed the *right* optic tract ; *c*. in 20 cases of hemianopsia with hemiplegia both on the same side.

61. Guérison de six aveugles-né par FIALA, Bukarest, 1878, 32 pages. (Bukarest, Thiel & Weiss.)

None of the six persons born blind had congenital ideas. They all had to learn. after the operation, how to direct the muscles of the eye. (All six were cases of congenital cataract in patients of 7–25 years of age and were operated successfully by discission.)

62. If in a certain minimum-intensity of light L=1 Sn 1 is recognized at 1′, Sn 2 will be recognized at the same distance with L<$\frac{1}{4}$, Sn 3 with L<$\frac{1}{9}$ etc. (ALBERTOTTI, *A. d'Ott*, 1878, 1, pag. 1–18).

63. The secretion of the aqueous humor is according to experiments upon animals made by CHABAS of Welna (*Pfluger's Arch.*, Bd. XVI, 2 and 3, pag. 143), proportional to the arterial pressure.

64. On the relation of the *pupils during sleep* with remarks on the innervation of the iris by E. RÆHLMANN and L. WITTKOWSKI at Strassburg, *Arch. f. Physiol.*, v. *E. du Bois-Reymond*, 1878. The size of the pupil does not only depend upon the incident light and the degree of accomodation, but also upon central influence which s regulated by the psychical and emotional influences from the outer world. The psychical centres of the cortex of the cerebrum as well as the various terminations of nerve of special sense are, during the normal conditions of waking, constantly in a certain state of medium irritation. Their action is propagated from the brain and spinal cord to the principal centres of the medulla oblongata, which transmit it through sympathetic channels to the iris with the tendency to dilate the pupil. The result of these

three components is the medium, more or less unstable equilibrium of the pupil. The pupil is narrow during sleep, because the irritation tending to dilate it is absent.

65 W. SANDER, *Arch. f. Phsych.* IX., 1. On the relation of the eyes to the waking and sleeping state of the brain.

66. There are *fibres tending to dilate the pupil, which do not come from the sympathetic nerve but directly from the brain*, and probably together with the fibres of one of the cerebral nerves, furnish the roots of the ophth. ganglion. (After experiments on cats by VULPIAN, *Gaz. Méd. de Paris*, 1878, No. 27.)

67. On the origin of the *nerves of accommodation* by V. HENSEN and C. VÖLCKERS of Kiel. (*A. f. O.*, Bd. XXIV, pag. 1–26, with plates, 1878.) The oculomotor nerve contains the nerve fib res for the accomodation and the narrowing of the pupil. The posterior portion of the floor of the third ventricle and the floor of the aquæductus can be irritated, the irritation of the different segments producing successively: 1. Contraction of the muscle of accommodation; 2. Of the pupil; 3. Of the rectus internus; 4. Of the rectus sup., levator palp., rectus inf., obliquus inf. (From experiments upon dogs.)

II.—THERAPEUTICS.

68. H. ADLER of Vienna, (*C. f. A.*, pag. 125, 1878), recommends *Vaseline*, (Paraffin-colloid from petroleum) as a constituent for eye-salves, since it neither irritates nor becomes rancid, [also *Seely*, *Higgens*, *Camuset*, etc.].

69. GOLDZIEHER of Budapest, (ibid., pag. 150), prescribes; Atropini 0.05 S. in aq. paux. Tere exact. c. *Vaseline* 5.00; especially for children, since it acts more quickly than the aqueous solution.

70. EMMERT, (*Correspondenzl. f. Schweizer Aerzte*, 1878, 7, Aug.), recommends *Vaseline* as a constituent for atropine, eserine, the yellow oxide and white precipitate of mercury, copper, alum, zinc.

71. SEELY of Cincinnati, (The *Clinic*, 1878, No. 13), recommends for trachoma an ointment of Hydrag. oxid. rubr., 0.60 *Vaseline* 30.00.

72. TWEEDY of London, found (with *Ringler*), that the extract of *Duboisia myoparoïdes*, (Scrophular, Australia), acts locally and generally *like atropine;* he used the solution of 1:20 also in the diseased human eye. Mydriasis sets in after 12–15 minutes, and disappears again after 24 hours.

73. v. HASNER, (*Prag. med. Wochenschr.*, 1878, No. 19), found the drug efficient in Iritis.

74. GERRARD of London, has prepared the alkaloid (*Duboisine*) which, in a solution of 1:120 produce mydriasis in 10 minutes.

75. According to GALEZOWSKI, (*soc. d. biol. de Paris*, March 30, 1878), this solution acts like one of atropine of 1:10 and can also be used in cases where atropine irritates.

76. This was also observed by v. WECKER, (*I. d. Therap.*, 1878, *Z. M. f.*, 1878), who ascribes to duboisine a stronger action than to atropine. Atropine remains valuable in iritis. In keratitis it seems to *v. Wecker* that "eserine will replace the atropine." Eserine can be instilled immediately after cataract extraction.

77. EXTRACT OF JABORANDI has only a mechanical action when locally applied to the eye. The instillation of one drop of the hydrochlor. of Pilocarpine (2.5 per cent.), produces a high degree of myosis and some spasm of accommodation; the subcutaneous injection of 1 *c. cm.* of the same solution, intense spasm of accommodation and slight myosis. (KÖNIGSHOFER, *Z. M.*, June, 1878).

78. ESERINE is efficient in keratitis and corneal ulcer, keratoconus, beginning sympathetic irritation and paresis of accommodation. (H. WILLIAMS of Boston, *Z. M.*, 1878, May).

79. DOR, (*sur le Chlorhydrat. de Pilocarpine*, Lyon, 1878), injects 0.02 dissolved in 0.2 Aq. daily or at greater intervals, in all three to twelve times in Irido choroditis or sympathetic Irido cyclitis.

80. SCHMIDT-RIMPLER of Marburg, (*Berl. Klin. W.*, 1878, No. 24), sometimes found improvement in chronic glaucoma by instillation of eserine (½ per cent. sol.). It has no specific action in keratitis. In prolapse of iris it is only indicated when the prolapse is peripheral; when central—atropine. Pilocarpine (1 *c. cm* of a 2 per cent. solution subcutaneously), has no specific action in Irido-choroiditis; injections of sublimate were sometimes more efficient.

81. SCHÖLER of Berlin, (1878, *Jahresb. d. Augenklinik für* 1877), has transplanted, successfully as to healing of a cornea which had been totally destroyed by ulceration, a piece of cornea 9½ *mm.* in diameter, taken by a trephine from the eye of a poodle. Sight was not restored.

82. v. HIPPEL of Königsberg, (*A. f. O.*, Bd. XXIV, 2, pag. 335–257), critically reviews the recent publications of *Schöler*,

Rosmini, *Dürr* and *Power*. He himself operates in deep narcosis and uses a trephine with a strong spring. The size of the piece is 4–5 *mm*. From the 5–21 day the inserted flap begins to become opaque.

83. The same reported at the Congress of Heidelberg on Keratoplasty, (*C. f. A.*, pag. 44, August, 1878).

84. SCHWEIGGER of Berlin, (*ibid.*) mentioned at the discussion, that he selected an especially favorable case of pure leucoma and transferred it to his assistant Dr. Sellerbeck for operation. A piece of human cornea (from an eye with pure retinal glioma), was taken out with the trephine and inserted into the centre. The piece of cornea remained clear for eight days, and the patient read with the eye large print; later it became opaque, fingers were counted at 2–3′. [In this case there existed in the remaining corneal tissue a tolerably transparent zone at the upper and inner margin. Fingers were counted at 3′ even 5 months after the operation].

85. SELLERBECK of Berlin, (*A. f. O.*, Bd. XXIV, 4, *Separatabdr.* 40 pages), on keratoplasty; the same case with remarks.

86. The antiseptic treatment in cataract extractions by ALFRED GRAEFE of Halle, (*A. f. O.*, Bd. XXIV, 1, pag. 232 and ff). The eye and lids are washed before the operation with a 2 per cent. solution of carbolic acid, and the eye kept covered until the operation with a sponge dipped into the same solution. During the operation the moistened sponge is used. After the operation the eye is again most scrupulously cleansed with the carbolized sponge. In the same instant when the sponge is removed from the eye the whole orbital region is closely covered by a piece of Lister's boric acid-lint, which has just again been saturated with a four per cent. solution of boric acid. Over this is spread a piece of the finest English oiled-silk which projects some millimeters over the lint on all sides, and which has also been dipped into the solution of boric acid. A uniform padding of cotton is then fastened to both eyes by a fine, elastic flannel bandage. This bandage is removed only once daily for the first three days. When the bandage is taken off, a sponge saturated in a solution of carbolic acid, must instantly be applied to the closed eye, in order not to expose the eye even for a moment to the influence of septic materials which may be suspended in the air. Among 114 extractions, performed at the Institute from the end of May until the middle of December, 1877, the loss was 2½ per cent., before

5–6 per cent. on the average. [Subsequent operations performed in the same manner, gave less favorable results. See Opthalmological review, *Arch. O. & O.*, VII. Knapp].

87. On moist salicylic bandages by HORNER, (*Mittheil. aus d. Ophth. Klin.*, 1878.) The cleansed eye is covered by a material which readily absorbs water, and this again by the cotton dipped into a solution of salicylic acid (1:300), finally a calico bandage Every 3–4 hours some of the solution is poured over; the bandage, which can remain 3–4 days, is kept clean and cold; *A*, prophylactic in lid-operations, enucleation; also in *cataract-operations; B*, in ulc. serp. corn. with or without lachrymal trouble and in the secreting kerato-conj. pust. scroful. of children.

88. The *antiseptic treatment in Ophthalmic surgery* according to HIRSCHBERG, (*C. f. A.*, 1878, pag. 151), is possible, but *not at all necessary* in cataract-extraction. Exact execution of the operation and absolute cleanliness are perfectly sufficient. Why has the antiseptic treatment not been recommended also for iridectomy?

89. JUST of Zittau. The boric acid bandage in ulcers corneæ serpens. (*C. f. A.*, Oct., 1878).

90. On peripheric capsulotomy by H. KNAPP, (*A. O. & O.*, VI, p. 545).

91. A *piece of iron* lay upon the anterior surface of the lens of a smith 41 years old. On approaching a strong *magnet* with two Grove's piles within 4″ from the eye, the splinter flew against the inner surface of the cornea and fell into the anterior chamber, whence it was removed with a piece of iris. Later, cataract developed and the lens was absorbed. HARDY of London, (*Lancet*, Bd. II, 13, 1878).

92. *A.* A foreign body slipped from the iris forceps; a pointed permanent magnet was introduced into the corneal wound and the foreign body immediately attracted. *B.* A miller showed, ¾ of an hour after the injury, a wound of the sclerocorneal margin 1‴ length. By a carefully introduced magnet the foreign body, which measured 1½‴ in length, was immediately drawn out. MCKEOWN of Ulster, (*British Med. J.*, May 4, 1878)

93. At the Ophthalmological Congress in Heidelberg, August, 1878, O. BECKER showed a patient from whom he had a year previously, extracted a cysticercus near the posterior pole of the eye through a scleral opening. Healing smooth; but partial detachment of the retina occurred.

94. The *extraction of a subretinal cysticercus*, situated at the upper

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part of the equator, was made by A. GRAEFE of Halle, (v. Kries, *A. f. O.*, Bd. XXIV, 1, pag. 148 and ff), after detaching the muscle by a meridional scleral section 1½ *cm.* long. One year later $S=\frac{3}{8}$, slight limitation of the visual field downwards. Retina attached. Formation of bonds in the vitreous.

95. *A subretinal cysticercus in the region of the posterior pole* was successfully removed by A. GRAEFE, (*A. f. O.*, Bd. XXIV, 1, pag. 209–232), after division of the rectus externus through a scleral section 5 *mm.* in length. (v. Graefe's knife was inserted at a distance of 19 *mm.* from the corneal margin). Fingers at 4′ eccentrically, even ½ year later. A series of cases were operated on in a similar manner. The eye is lost, if left alone. "The operation offers much better chances—even in the cases where the parasite is in the region of the posterior pole, which cases A. v. Graefe, even in 1868, regarded as excluded from all attempts at removal."

96. On extraction of a subretinal cysticercus with preservation of sight by H. COHN of Breslau, (*C. f. A.*, 1878, pag. 146.) In a female, a subretinal cysticercus of 7 *P.* was seen down and inwards from the optic disc. Without narcosis the ext. rect. muscle was divided, the eye rotated inwards; the cataract knife was inserted 25 *mm.* from the corneal margin and from here the sclera cut forwards, layer after layer, until the section perforated to the extent of 9 *mm.*; the worm was extracted by an iris-forceps; section of muscle and conjunctiva united by sutures. Healing good. Sn 2, visual field normal except an angular defect upwards. Later, two other cases were extracted with the same success.

97. A. GRAEFE, (*A. f. O.*, Bd. XXIV, 3), reports the favorable results of 12 *cases later operated upon by him in a similar manner within a short time.*

98. SCHÖLER of Berlin, delivered a lecture on *Enucleation of the Eye*, on January 21, 1878, (*Berlin. Klin. Wochenschr.* No. 16, and *Jahresbericht von Schöler's Augenklinik f.* 1877). All the indications for the enucleation of the eyeball—apart from the malignant neoplasm—would be fulfilled by cutting the optic nerve with all the ciliary nerves which enter the sclera very near it. (*Neurotomia optico-ciliaris*). The tendon of the rectus externus is divided, the globe dissected with a small pair of scissors, the optic nerve divided, the globe rolled inward as much as possible, all the fibres which have perhaps remained, are then divided by a small tenotome muscle and conjunctiva united by sutures. Schöler has performed

10 operations of this kind.* He later reported (*Berl. Klin. W.*, 1878, No. 45) on 30 operations.

99. HIRSCHBERG, (*C. f. A.*, 1878, 133, with appendix), has performed the operation six times, four times in slight cases which came for prothesis; once in severe and sudden spontaneous cyclitis in an eye blind for years from injury. He divided the internal or external rectus, simply dissected up by enucleation-scissors and forceps the optic nerve entrance, turned the posterior pole of the eye forward and liberated it from all adhering fibres; he then re-implanted the half enucleated globe, and, of course, united the muscle by a suture.

100. SCHWEIGGER, (*Berl. Klin. W.*, 1878, pag. 20), operates always from the internal rectus muscle. He is convinced that the neurotomia optico-ciliaris produces the same effect as the enucleation, but *no more*. Where sympathetic iridocyclitis has once manifested itself, the enucleation is perfectly useless. The results of the prophylactic enucleation are beyond our critique. But everything indicates that the operation should be done as early as possible, when the conditions for the development of sympathetic ophthalmia are once present. About 20 cases, operated on in Schweigger's wards. Inaugural Diss., of BEHRING. Berlin, Aug. 15, 1878.

(The third and fourth parts of this report will appear in the next number).

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* In regard to the history of this question neither Boucheron or Vignaux are of any importance; the former has not operated, the latter has verbally translated a passage from Mooren without quoting his authority. v. Graefe first recommended the division of the optic nerve, (also the intraocular division of ciliary nerves), instead of enucleation in sympathetic irritations. (*A. f. O.*, Bd. III, 2, pag. 452). "I have recommended in similar cases, (viz., sympathetic inflammation), to divide the optic nerve instead of extirpating the eyeball. This would eventually have the advantage of preserving the globe. This proposition has been first executed, as far as I know, by Dr. A. Weber of Darmstadt." And further: "I have formerly recommended the division of the optic nerve instead of the enucleation, where sympathetic opthalmia was threatening." (A. v. Graefe, *Verh. d. Berl. med. Ges.*, von 19 Juli 1867). MOOREN in his monograph of 1869, says: "A second proposition of Graefe to divide the optic nerve trunk, is based essentially upon the above-mentioned principle; for the famous author had intended by it a division of the ciliary nerves. This operation, it seems, has not been executed by ophthalmologists, except by A. Weber." Rheindorf has operated upon one case in 1865 successfully, (*De l'Ophth. symp., pag.* 26), and Landsberg one case without success in 1869.